A SYSTEM OF MEDICINE.

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EDITED BY

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VOLUME THE THIRD,

CONTAINING

LOCAL DISEASES

(CONTINUED).

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NOTE BY THE EDITOR.

It has been found desirable to extend this "System of Medicine" to four volumes, and to publish in this, the third volume, those articles which complete the section on "Diseases of the Digestive System," and those which contain an account of "Diseases of the Respiratory System."

The delay which has occurred in the appearance of the present volume is due to the fact that the Editor did not receive, many months after he expected to do so, the MSS. of those articles which were necessary to complete the section on "Diseases of the Circulatory System," several of the papers on which subjects had been for a long time printed. A short time ago, therefore, in order to avoid further postponement, he decided to change the order of sequence originally intended, and to publish first the section on "Diseases of the Respiratory System."

The diseases of the circulatory system, of the blood-glandular, of the urinary, the reproductive, and cutaneous systems, will constitute the fourth volume.

In dealing with so large a series of subjects as those which make up the section on "Diseases of the Respiratory System," some repetition of statement and occasional diver-

gence of opinion have been found inevitable. The Editor has, however, thought it desirable to allow the occurrence of the former, in order to give completeness to separate articles, and has taken pleasure in the representation of the latter, inasmuch as, in his opinion, such divergence expresses, with the greatest faithfulness, the present state of scientific knowledge on many unsettled problems of pathology, and by so doing will prove more useful than would any attempt at enforced uniformity of teaching.

The general doctrine of Tubercle; the relation which that material bears to local and general diseases; the precise meaning of certain morbid conditions, the characters of which are matters of familiar recognition; and the inter-relations of many well-known words, are each and all of them susceptible of various interpretations: and the Editor is grateful and glad to be able to bring together in a connected form, under the notice of his professional brethren, the views that are severally entertained by those distinguished authors who have already shed much light upon these obscure regions, and have furnished many of the results of their finest labours in the present work.

<div align="right">J. RUSSELL REYNOLDS.</div>

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ERRATA.

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DISEASES OF THE MOUTH.

By Charles E. Squarey, M.B. Lond.

1. Hæmorrhage from the Mouth.

The mucous membrane of the mouth is much less liable to hæmorrhage than is that of the nostrils; the most frequent seat of hæmorrhage is the gums.

When not profuse, the symptom complained of is a continual spitting of blood; if profuse, it may run from the mouth in a stream, or it may be swallowed, and cause vomiting with symptoms resembling hæmorrhage from the stomach; this occurs more frequently in children than in adults. Again, hæmorrhage, coming from the back part of the mouth, may trickle down into the larynx and cause irritation and cough, with expectoration of blood, resembling pulmonary disease.

The diagnosis is, however, generally very easy immediately that attention is directed to the mouth, when the bleeding point may be at once detected.

If the hæmorrhage be too profuse for the mouth to be inspected, the patient's head should be held forward to allow the blood to run out; and if it does so without coughing or vomiting, and if it is of a bright red colour, neither frothy nor grumous, there can be no hesitation as to its having its origin in some part of the mouth or pharynx. If the bleeding is not very profuse, and no bleeding point can be seen, if the patient feels a tickling sensation in his throat before he expectorates, or if he has a constant disposition to deglutition, and has no difficulty in hawking up the bloody sputa, it may be inferred that the blood proceeds from some part of the fauces; and in such cases, on inspection, the pharynx will be seen to be more or less congested, and the vessels to be full and very distinct. Again, when hæmorrhage comes from this part, the use of a slightly astringent gargle, and even the mere act of swallowing solids, will generally cause a sense of discomfort if not of actual pain.

The spitting of blood is almost always referred by patients to the lungs, and in some cases it is difficult at first to decide whether it does come from the chest or not. Amongst the most puzzling are those cases where the patient every morning coughs up a little blood;

more or less bleeding has taken place during sleep, and has collected about the pharynx and larynx, and in the morning, on clearing his throat, he hawks or coughs up this blood.

The inspection of the pharynx, the history, and the absence of all pectoral symptoms will at any rate, in a short time, if not at once, reveal its true origin. The appearance of the blood, too, will also aid in the diagnosis. Usually, when expectorated from the lungs, it is liquid, florid, and more or less frothy, owing to its admixture with air in the respiratory passages. When from the mouth, it is only very slightly frothy from the admixture with the saliva, and more frequently, especially in those cases when it is hawked up every morning, it consists almost entirely of little coagulated masses of blood in a clear saliva.

In all difficult cases the patient should be made to rinse his mouth out with lukewarm water before inspection.

The CAUSES of this affection are those of hæmorrhages generally, such as plethora, the hæmorrhagic diathesis, scurvy, purpura, and the like. The local causes are violence, blows of any kind, biting the tongue, an epileptic fit, extraction of teeth, the presence of inflammation or of ulceration. Wood records a case of very serious hæmorrhage from the lancing of a vascular tumour of the palate in mistake for an abscess.

When the "hæmorrhagic diathesis" is present, most serious losses of blood may take place, and in the worst of these cases it oozes from all points of the gums and palate. In such conditions the smallest wound or the least blow is sufficient to give rise to the most serious hæmorrhage.

When bleeding from the mouth is vicarious, taking the place of hæmorrhoidal or catamenial discharge, it is generally periodical; and when it occurs from inflammation or ulceration, as in the mercurial sore mouth, the hæmorrhage may come from the surface of an ulcer, but more usually it is from the general surface of the mucous membrane.

In many cases of serious bleeding from the mouth Frank has observed the veins of the part to be in a varicose condition; Vogel has also noted this condition, and has given to it the name of "Hæmorrhoides Oris."

The PROGNOSIS depends entirely on the constitutional condition of the patient, the quantity of blood lost, and the effects that its loss may have produced.

TREATMENT must be guided by the cause. If the hæmorrhage be simply due to violence, the patient should be given ice to suck; and should use astringent washes or powders, such as alum, kino, catechu, and tannic acid; with pressure if necessary. If the hæmorrhage be due to a general constitutional disease, remedies to correct that con-

dition should be administered; but if it takes place vicariously of natural discharges, efforts should be made to restore those functions which have been impaired.

DIFFERENT FORMS OF INFLAMMATION OF THE MUCOUS MEMBRANE OF THE MOUTH.

The form assumed by Inflammation of the Mucous Membrane of the Mouth depends partly on the nature of its cause, and partly on the constitutional condition of the patient.

Inflammation is much more frequent in children than in adults, and it may be either a primary, or a secondary or sympathetic, affection.

2. STOMATITIS SIMPLEX.

This is known also as Simple Erythema of the Mouth, or Common Diffuse Inflammation of the Mouth.

It is a slight and badly-defined disease, lasting rarely more than a few days if properly treated, and causing very slight constitutional symptoms. It may, however, end in ulceration if not attended to. It is most common in infants between two months and one year old, and seems to be due entirely to bad feeding and bad hygienic conditions. It is thus much more frequent amongst the children of the poor, especially in those brought up by hand, than in those of the upper classes. It is, however, sometimes the result of sore or inflamed nipples of the nurse.

The symptoms first drawing attention to the disease are the general restlessness and peevishness of the child, loss of appetite, dryness of the mouth, and now and then some slight diarrhœa, with more or less distension of the abdomen.

On looking into the mouth, the mucous membrane is seen in places to be redder than natural, raised above the general surface, and presenting a dryish, glistening appearance.

These patches are generally to be seen on the inside of the cheek, about the level of the line of junction of the gums, and round about the angles of the mouth; they are irregular in shape and size; the larger patches are formed by the junction of the smaller ones, and by their thus joining together they may extend so as to cover the whole of the mucous membrane of the mouth, and even of the palate and the gums. Such extensive inflammation is, however, exceedingly rare. The centres of these patches have a yellowish tinge, their margins are bright red, and there may be even some slight effusion of lymph, forming a soft membrane on the surface.

Simple Stomatitis easily gives way to simple remedies, such as a dose of castor oil and the regulation of the diet; but if attended with gastric irritation—to which it frequently is secondary—small doses of

magnesia, or of rhubarb and soda, should be given. If the child is being brought up by hand, the addition of lime-water to its milk, in the proportion of one part of lime-water to two parts of milk, will be of service.

The mouth should always after every meal be washed out by dipping a soft rag in lukewarm water and gently rubbing it over the internal surface of the mouth.

When due to hot or acrid substances taken into the mouth, mucilaginous drinks should be given; if due to teething, the gums, if necessary, should be lanced.

3. THRUSH.

THRUSH—known also as Diffuse Inflammation of the Mouth with Curdy Exudation, as White Mouth, or Le Muguet—is a form of inflammation of the mouth met with at all ages, but much more frequently in new-born infants and children than in adults. It occurs usually as a complication of other diseases.

In new-born infants it is, however, so easily produced that it has been called in them *idiopathic;* but as it is invariably preceded by some gastric symptoms, and more or less diarrhœa, it is in infants, as in adults, a secondary affection. The change in the secretion of the mouth from an alkaline to an acid reaction is favourable to the production of this disease; and as during the first weeks of infancy the mucous membrane of the mouth yields, even in a state of health,—and wholly independently of whether or no the child is brought up at the breast,—an acid reaction, this circumstance accounts for the far greater liability of infants than of adults to this affection.—(West.)

The invasion of this disease is generally marked by more or less constitutional disturbance; the symptoms first noticed by the mother being slight feverishness, irritability, loss of appetite, and the presence of a little diarrhœa: severe diarrhœa is not by any means a constant primary symptom. The mouth is generally hot and dry, and the secretion of the saliva lessened.

On looking into the mouth, if the child be seen before the characteristic white curdy exudation has made its appearance, the mucous membrane is found in places to present the same appearance as that described in Simple Erythema. Small red raised patches are to be seen. They may appear first either on the tongue, the insides of the cheeks, or at the inner margin of the lips, at the angles of the mouth, and generally at this latter part first. In this stage it cannot be diagnosed from simple erythema, the heat and dryness of the mouth and the red raised patches being similar in both; but it is rare in this disease for the child to be brought under notice so early that some of the characteristic curdy exudation is not to be seen on some part of the mouth. The time that elapses between the first symptom of the

disease and the appearance of the curdy exudation has not been determined. Wood says two or three days, or even less.

This curdy exudation commences in these inflamed patches in isolated points, which approach each other gradually, and in certain cases form a membrane altogether continuous. When this happens, the inflammation is very intense. There is scarcely any part of the mouth anterior to the palatine arch which may not be covered with this perfectly continuous false-membranous product; and it offers a surface as if covered with a croupy or diphtheritic exudation; hence the use of the term Diphtheria of the Mouth, which by some authors has been applied to this affection, a name which, from the severity of the disease to which it refers, should, I think, never be used for an affection such as Thrush, as it is undoubtedly liable to cause much anxiety amongst the friends of the little patient.

From whatever cause produced, the false membrane is generally seen in patches, altogether or partially isolated, and thickest on the inner sides of the cheeks and mouth, where they could be easily confounded with curds of milk, so much do they resemble them. On the tongue, the roof of the mouth, the pharynx, and in the œsophagus, the patches of false membrane are more irregular in shape and size.

At first this exudation is rather firmly adherent, and cannot be raised without causing slight bleeding; but, after a few days, it becomes loose, falls off, and is replaced by fresh exudation. Its colour is usually white; sometimes it assumes a yellowish or brownish colour: the latter is considered an unfavourable sign.

The mouth of the infant is hot, and frequently the nipple of the nurse is more or less excoriated.

More or less diarrhœa is generally present; the stools are fœtid, and of a greenish yellow colour; sometimes white curdy masses are passed with the stools—and this is considered evidence of the disease having extended to the bowels, though I am not aware that this has been proved by the microscopic discovery in these masses of the cryptogamic growth peculiar to the exudation in the mouth. In two or three cases in which I examined these white masses, no trace of it was to be seen; the masses seemed to consist entirely of curdled milk.

Redness around the anus and on the buttocks is not uncommon, and is probably due to the acrid discharges. Valleix, however, states that he has found Thrush commencing with erythema of the buttocks, rapidly followed by diarrhœa, and swelling with redness of the papillæ of the tongue, which, a few days after the occurrence of the first symptoms, were covered with the characteristic exudation.

The affection may be so slight as to be considered entirely local (idiopathic), but in by far the greater number of cases there is some acceleration of the pulse and febrile heat.

As it usually occurs the disease is without danger; but in weakly, scrofulous children, when secondary to gastric and intestinal disease, it often proves fatal: but in these cases death is caused by

the general condition, of which Thrush is merely the local expression, and not by the Thrush itself.

The duration of Thrush is uncertain, sometimes lasting only for three or four days, at other times becoming chronic, and lasting for several weeks; it often returns after apparent cure, and this may happen repeatedly during a period of several months.

Its appearance in the course of acute and chronic diseases is a sign of grave importance, especially in the latter, it invariably in these cases foretelling a not far-off fatal termination.

In the acute specific diseases, and other acute attacks, although its appearance bodes no good, yet, as I have frequently heard Sir William Jenner say, it is not nearly of such grave import as in chronic cases, and I think I am not wrong in stating that in these acute diseases it does not interfere with recovery.

It may occur in all diseases which undermine the general constitution, especially those in which the alimentary canal is affected. Anæmia in delicate girls is, in rare cases, sufficient to cause it, as in the case of a girl, aged 19, under my care last year.

The peculiar exudation of Thrush has been seen to extend from the mouth down the œsophagus to the stomach : its appearance in the stomach is, however, rare. Valleix states once in twenty-two cases. It has also been seen in the small intestine, where it covered one of Peyer's patches, and once in the large intestine, in the cæcum.

Under the microscope, the curdy exudation of Thrush has been found to consist of thickened epithelium cells, mingled with numerous minute cryptogamic sporules or seeds, from the midst of clusters of which long, thread like, jointed, and branching plants arise intertwining with one another. It was discovered by Berg of Stockholm and Grubz of Vienna at the same time, and has been named the Oidium albicans.

ETIOLOGY.—It has been, I believe, now undoubtedly proved by Berg, that this plant is the origin of the disease ; the sporules, floating about in the respired air, attach themselves to the mucous surface and, under favourable circumstances, become developed and propagate.

These circumstances may consist in some previous morbid state, changing the buccal secretion from its normal alkaline character to that of acidity, which probably favours the growth of this as of other microscopic fungi.

The complaint is contagious, as proved by Berg, who in four instances, by transplanting the plant from a diseased to a healthy child, succeeded in propagating the disease.

Of the predisposing causes, it may be said generally, that whatever tends to impare the vital powers of the child predisposes to this affection : thus it is found chiefly amongst the children of the poor, whose surroundings in large towns are generally anything but healthy.

It is especially noticed in children brought up by hand, in children prematurely born, and in those nursed by unhealthy mothers. Inflamed and irritated nipples will also produce it.

Boerhave says in some cases it is due to protracted suckling; and this, perhaps, explains its presence sometimes in children well-fed, and brought up, apparently, in the most favourable circumstances.

Barthez and Rilliet think that in some mild cases, in which they have met with it in children of primiparous women, it has been due to the fatiguing effort of suckling owing to the nipples of the breast being little formed. Dr. Strœblin of Geneva has also noted this cause.

TREATMENT.—In mild cases, when there is not much constitutional disturbance, very little is required beyond local applications. A dose of castor oil to free the stomach and intestines from any irritating matters that may be present, a careful regulation of the diet, and the local application of a little borax, either dissolved in honey, or in the form of powders, mixed with sugar in equal parts, is all that is necessary.

If the child is badly nourished, and the attack is attended with vomiting and purging, the treatment may with good effect be still commenced with castor oil. If the purging still continues, magnesia and chalk, with a little opium, should be given occasionally.

If the child is being brought up by hand, lime-water should be added to its milk in the proportion of one part to three of milk : if at the breast, the nurse should be examined to see that she is quite healthy, that the nipples are not excoriated or inflamed in any way, that the milk is good, and that there is plenty of it; and she should be advised to give up all acescent foods.

The child's mouth should be washed out after every meal, as directed in Simple Erythema.

Cod-liver oil in doses of ʒss., three times a day, may also be given, and if retained, it does much good. In severe cases, if the breath be fœtid, and if there be much swelling of the lips and gums, chlorate of potash should be given freely in doses of from three to five grains dissolved in a tablespoonful of water, every four hours ; and in cases inclined to be chronic this makes a very good wash for the mouth.

Creasote, vinegar, and alcohol have been recommended as local applications. A solution of sulphate of zinc, two grains or more to the ounce, may also be used in chronic cases.

Sir W. Jenner recommends a solution of sulphite of soda, in the proportion of ʒj. to the f. ℥j., and ascribes its beneficial effect to the destructive influence of the sulphurous acid on the parasitic fungus.

Whatever washes may be used, the application should be made from four to five times a day, and care should be taken not to irritate the inflamed parts by rubbing them more than is necessary.

In adults the same applications may be made. Dr. Tanner recommends a solution of bromide of soda, ʒj. to the f. ℥j., with a little glycerine. The mouth should be frequently rinsed with this.

4. Ulcerative Stomatitis.

In the two previous diseases of the mouth no ulceration or loss of substance occurs; but the disease we are now about to consider is characterised by the formation of thick, adherent, yellow patches of membrane, and by inflammation, erosion, and ulceration of the subjacent parts. Cancrum oris has by some authors been described under this head, but in this work it will be treated as a distinct disease, inasmuch as it differs entirely in the character of its ulcer.

The membranous form of this disease is most probably the early stage of the ulcerative affection; it may not, however, proceed so far as ulceration, the thickened patch of membrane being either absorbed or thrown off without the formation of an ulcer.

The patches of membrane are at first white; they then become grey, and sometimes almost black; the mucous membrane around them is red, swollen, and inflamed; the false membrane looks as if imbedded in it; it is firmly adherent at first, and, if raised at this time, is found to have covered a superficially eroded, bleeding surface.

If it proceeds to ulceration, the membrane comes off, and discloses an ulcer with irregular red or violet-coloured margins, and generally covered with more or less of a thick, yellowish, pultaceous exudation.

The onset of the disease is not marked by any special symptoms, and it is rare for the child to be brought under notice till more or less ulceration exists.

On opening the mouth, irregularly-shaped, isolated patches of ulceration are to be seen on the inner sides of the cheeks opposite to the line of junction of the teeth, and extending down from this below the angle of the mouth to the gums of the incisor and canine teeth; the gums are swollen and spongy, and bleed easily; later on they become retracted, the teeth are laid bare, and become loose.

The ulcers, at first more or less isolated, soon run together, and form a line as above described, with irregular, greyish, and raised edges, the membrane around being swollen and inflamed, and of a bright red colour. The inflammation may spread rapidly, and involve the whole mucous membrane of the mouth, and even of the palate; but such severe cases as these are exceedingly rare. At first the ulcers are quite superficial, but if left to themselves they become deeper, and the disease gets into a chronic state which is sometimes very difficult to cure.

For some time after the ulcers have healed there is more or less redness of the new-formed membrane, and the subjacent tissue remains hard and thickened.

Accompanying the ulceration there is more or less swelling of the side of the face attacked; and the neighbouring glands, sublingual and submaxillary, become hard, painful, and very tender to the touch. The surrounding cellular tissue very rarely participates in this inflammation.

The breath in bad cases is generally intensely fœtid, and the flow of saliva much increased.

The disease generally commences about the gums,—the lower ones more frequently than the upper,—and in by far the greater number of cases only one side of the face is attacked, the left side being the most frequently favoured in this respect.

It is not a dangerous disease, thus differing greatly from cancrum oris, which is very fatal. It may be easily distinguished by the fact that, in this disease, the swelling of the face is neither hard, resisting, nor circumscribed, and the skin neither tense, shining, nor hot, symptoms which are always present in cases of cancrum oris. The characters of the ulcer are also very different. See Cancrum Oris, p. 15.

DURATION.—If not treated it may last some months, and even under treatment, if the child be living under bad hygienic conditions, it is sometimes very troublesome to cure. It has a great tendency to recur.

AGE.—It is most common between the ages of five and ten years; it may come on during weaning, and it is said to occur more frequently in boys than in girls, though my experience points entirely in the other direction. It is more common in autumn than at other times of the year.

CAUSES.—These may be local or constitutional. Thus, caries of a tooth, or fracture or necrosis of the bones of the jaw, will produce it in weakly, scrofulous children; but it is essentially one of those diseases which result from want of proper care and nourishment, and is almost entirely confined to the children of the poor, who are badly looked after, badly lodged, and badly nourished, and living in low, damp, and deficiently ventilated houses.

The TREATMENT consists of constitutional and local remedies. The child should be removed, if possible, from the bad hygienic conditions under which it may have been placed, and tonics, as steel wine and cod-liver oil, should be administered.

The secretions should be attended to, and kept regular and natural.

Of the topical applications, powdered alum, nitrate of silver, nitrate of mercury, and hydrochloric acid may be named, and of these alum, either used as a powder or in the form of a wash, will generally be found quite sufficient; but if the ulcers be slow to heal, their surface should every now and then be cauterized with nitrate of silver. Gargles of chlorate of potash, from five grains to one scruple to the ounce, or even stronger, may be used, other means failing; its internal administration also seems in some cases to have a very

beneficial effect. In one obstinate case I found brushing over the raw surface with a mixture of equal parts of the tincture of the sesqui-chloride of iron and glycerine to have a very good effect.

GINGIVITIS is an affection resembling ulcerative stomatitis, but differing from it in its seat. In this disease the gums alone are affected; they become swollen, spongy, bleeding, and ulcerated round the margins of the teeth; they retract from the teeth, and render them so loose that they may fall out.

It is common at the same ages as ulcerative stomatitis, and is produced and predisposed to by the same causes. The treatment is exactly the same.

5. APHTHOUS STOMATITIS, OR FOLLICULAR STOMATITIS.

The word Aphthæ, derived from the Greek ἅπτω, to inflame, was formerly applied to all the various inflammations of the mucous membrane of the mouth; but now it is used in a much more restricted sense, being applied only to all those small isolated ulcers so frequently seen on the mucous membrane of the mouth.

Follicular Stomatitis I have also placed with this disease, it being considered by some, and by Bellard especially, as the origin of all aphthæ.

Follicular inflammation may not go so far as to cause ulceration; the follicles then are seen to be inflamed independently of the surrounding mucous membrane. They appear as small, red, slightly elevated, round spots, hard and almost shotty to the touch, with, in the centre, a little black punctum, the opening of the follicle. If at this stage the inflammation does not subside, the hard central part softens and ulceration takes place; the ulcer being round, having sharply defined edges, surrounded by a small circle of inflammation. If many follicles are inflamed close together, the ulcers may join together, forming irregularly ulcerated patches.

The most frequent source of aphthæ, however, is a vesicular eruption, very much resembling herpes, which appears on any part of the mucous membrane of the mouth, and is not confined to the follicles.

In the first stage small vesicles are seen, containing in the beginning a clear limpid fluid, which soon, however, becomes whitish, surrounded by a slight circle of inflammation. The vesicle becomes distended, and finally ruptures, leaving in its place a small ulcer, with a greyish yellow base, and having a bright red and sharply defined regular margin. The ulcer is often very painful, sometimes so much so as to cause the patient to refuse as much as possible all nourishment. The buccal secretions are always more or less increased, but abundant salivation is an exceptional symptom.

Generally about six or seven of these ulcers are to be seen on various parts of the mouth; it is very rare to find only one, and still

rarer to find those that do exist running together and becoming confluent. I have never seen a case of confluent aphthæ.

The most frequent seat of these aphthous ulcers is the internal surface of the lower jaw, in the sinus, between the lips and the gums then on the sides of the tongue. They are occasionally to be seen on the tonsils and palate, but are less frequent in this position than elsewhere.

There is generally more or less inflammation of the gums, especially when the aphthæ are in the sinus, between the gum and the buccal wall.

Very little constitutional disturbance accompanies this disease when occurring in adults; it is almost entirely local, the pain and discomfort in the mouth produced by the ulcers being the only sign of the disease. In these cases, when the medical man is called in, the disease has always existed for some few days, and has become thoroughly confirmed.

In severe cases in children there may be much fever, great difficulty in swallowing, great pain and swelling, and very fœtid breath; but such cases as these are very rare; it is quite exceptional for the constitutional symptoms to have any marked importance.

Ordinarily the little patients are slightly feverish, restless, and irritable, with white tongues, no appetite, and some thirst. Under judicious treatment all these symptoms rapidly disappear, and the ulcer quickly heals.

The disease generally lasts about a week, sometimes longer, especially if successive eruptions come out. It is common at all ages, attacking equally children and adults. It is very liable to return.

Simple aphthæ do not constitute a dangerous disease; but the confluent variety may be accompanied with much constitutional disturbance, with vomiting, diarrhœa, &c., which show its propagation to other parts of the intestinal tube, and in this form it may prove fatal. It is very common in some countries, especially in Holland, and it attacks people in ill-health, women in childbed, and those badly nourished more frequently than others.

CAUSE.—No definite cause is known. It is observed to be more frequent in weakly children, and those subject to catarrhal and eruptive diseases. Unfavourable hygienic conditions predispose to it; and yet, again, it is occasionally observed in children placed in this respect in the most favourable conditions.

Barthez and Rilliet have had occasion to think that in some cases it must have been hereditary.

TREATMENT.—In by far the greater number of cases very little medicinal treatment is required. The patient should be placed in as favourable hygienic conditions as possible; the diet should be regulated, and should consist of easily digestible food; all irritating matter should be excluded. Wine, if the child be low, should be given in moderate

quantities, and the secretions should be seen to be healthy in character, and normal in amount.

If purgatives are required, a small dose of rhubarb and soda, or, in an adult, a dose of calomel, should be given; and in rare cases, if there be swelling of the face, with much fever and a strong pulse, the application of two or three leeches will be found useful. Tonics, however, are much more frequently required than lowering measures.

The local treatment consists in the use of washes for the mouth—at first, whilst there is much inflammation—of a demulcent character, as linseed tea, mucilage, &c., followed, on the subsidence of the inflammation, by astringent washes, as solutions of alum, acetate of lead, sulphate of copper or zinc,—and these should be painted on the ulcers with a camel's hair brush.

Nitrate of silver applied in the solid form is also very effective.

6. Gangrenous Stomatitis.

Synonyms.—Gangrenous Inflammation of the Mouth; Gangræna Oris; Cancrum Oris; Sloughing Phagedæna of the Mouth; Necrosis Infantilis.

This is by far the most serious of all inflammations of the mouth. Formerly it was considered as only an aggravated form of the ulcerative stomatitis previously described, but its peculiar characters and almost invariable fatality have induced all modern authors to describe it as a distinct disease.

Fortunately it is almost as rare as it is fatal. It invariably attacks either very delicate, badly nourished, scrofulous children, or those whose health has been previously much undermined by some severe disease, especially such diseases as are connected with important changes in the circulating fluid, as the acute specific fevers.

Symptoms.—The disease is very insidious in its origin, being accompanied with scarcely any pain in its early stages; and from this, and from the fact that the child is generally suffering from some other serious disease, or just convalescing from it, it may not be discovered till it has made some considerable progress.

There is some difference of opinion amongst authors with regard to its commencement. Barthez and Rilliet assert that it always begins by ulceration in the mucous membrane, whilst others, as Bellard and Richter, believe that it commences in the substance of the cheek by swelling and the formation of a central hard spot or nucleus of infiltrated and indurated fatty tissue, surrounded by a tense elastic, but less firm, swelling, passing off into the texture of the adjacent parts; and that the ulceration of the mucous membrane is secondary to this.

The first symptom of the disease which may attract notice may be either swelling of the cheek, fœtor of breath, or profuse salivation. It is noticed almost invariably by the mother or nurse first, the

little patient rarely being rendered sufficiently uncomfortable by it as to complain of it. Thus it is a complication which should always be thought of and looked out for in the convalescence of weakly children from severe diseases, especially when these occur between two and five years of age, this being the period at which cancrum oris is most common.

Swelling of the face is the symptom generally noticed first, and its character is so peculiar that it may be almost considered as pathognomonic of the disease.

The skin has the appearance of being tightly stretched over the swelling; it is red and shining, with a bright red spot in the centre, shading off gradually into the natural colour of the cheek. It feels dense, hard, and distinctly circumscribed under the bright red central spot, and to the hand it is perceptibly hotter than the other side of the face. There is rarely much tenderness, if any, either on pressure, or movement of the jaws; the breath has invariably at this early period a distinctly gangrenous odour, and the secretion of saliva is greatly increased.

On examining the mucous membrane of the mouth, there is seen on some part of its internal surface, it may be either on the inner surface of the cheek, at the line of juncture of the teeth, or on the gums, or the sulcus between the gums and buccal wall,—and Barthez and Rilliet have found the sulcus between the lower gum and the buccal wall the most frequent spot,—a small ulcer of an irregular shape, with more or less jagged edges, and having a dirty brownish slough attached to it. The edges are at first a bright or livid red colour, and bleed easily. The ulcer spreads very rapidly, and communicates the same condition to the parts which are in contact with it, so that from the gum it passes to the buccal wall and *vice versa.* If in the sulcus, it spreads at once throughout its length, and upon both sides. The teeth, very quickly denuded of the gums and covered with putrefying matter, soon become loose, and may fall out, and further on in the disease necrosis of the maxillary bones takes place. Taupin affirms that he has even observed separation of the necrosed parts, but this is very rare, as death generally occurs too soon for them to be thrown off.

The saliva, very much increased, is fœtid, and soon becomes discoloured with the discharges from the ulcer.

Coincident with the extension of the ulceration in the mouth, changes take place in the external swelling; the bright red central spot, increasing somewhat in size, gradually becomes more and more livid, till at last in the centre it is quite black. At first very small, this black spot increases rapidly, till it occupies the whole portion of the cheek, covering the hard central nucleus; and then the process of separation commences; the slough is thrown off, if the child lives long enough, and the cavity of the mouth is laid open.

The slough generally makes its appearance between the third and seventh days of the disease. After its separation the ulceration still

continues advancing into the remaining portion of the cheek, gradually eating it away, and producing the most horrible appearance, till at last death closes the scene.

With all this ulceration there is rarely much pain, and sometimes, as Dr. West says, the cheerfulness of the child is undisturbed, and it will sit up in bed playing with its toys till long after the appearance of the black eschar in the cheek has shown the case to be almost hopeless. But it is only rarely, and in mild cases, that so few constitutional symptoms are present. Generally, although not complaining of pain, the little patient is more or less prostrated, taking no notice of whatever may be going on, and having a great objection to be disturbed. Sometimes there is much drowsiness, the child almost continually sleeping. Much of this condition, however, may be due not so much to the disease itself as to the state to which the child had been reduced by its previous illness. Food and nourishment is invariably taken quite well, even up to a few hours before death.

The glands in the neighbourhood are generally much swollen and hard, and there is much infiltration of the cellular tissue around.

After the separation of the slough, however, the child may recover. The ulceration then takes on a healthy form, granulations are thrown out on all sides, the swelling disappears, and the hole is gradually filled up, though always with more or less retraction of the cheek and deformity of the face. In some cases a small fistulous opening may remain; in other cases, if the ulceration has been extensive on the contiguous surfaces of the cheek and gums, these may be united in the process of healing, causing great deformity, and diminishing very much the little patient's power of opening its mouth.

The pulse depends, like the rest of the constitutional symptoms, on the previous condition of the child. In some cases noted by Barthez and Rilliet, when the gangrene of the mouth was the principal symptom, it was never higher than 120, and gradually became slower and weaker as the disease progressed towards a fatal termination.

The amount of fever that is present has never, so far as I know, been determined by the thermometer. The side of the face attacked is invariably hotter than the other; but the temperature of the body, in judging by the hand the cases that I have seen—is not at all elevated. The skin is generally more or less dry. There is rarely any sweating.

The tongue is moist throughout, with more or less of a dirty yellowish fur on the dorsum. The digestive system seems to be very slightly impaired in this disease, all nourishment being taken at first with avidity, and even at the end without any reluctance.

At the post-mortem examinations there are invariably found some small nodules of pneumonia scattered throughout the lungs. Lung symptoms are, however, not noticed during life.

PROGNOSIS in this disease is always very grave; in a large majority of cases it terminates fatally. Of twenty-one cases under the

care of Drs. Barthez and Rilliet, twenty died ; of ten under the care of Dr. West, eight died ; and of three cases that I saw whilst at the London Fever Hospital, two died.

PATHOLOGY.—Gangrene of the mouth invades all the tissues forming the buccal wall. The conditions of the skin, mucous membrane, teeth, and bones have been fully described amongst the symptoms of the disease. Of the intervening tissues, the fatty cellular tissue and the muscular tissue are found to be infiltrated with serosity, and certain parts, corresponding to the extent of the ulcer, mortify and are thrown off with the slough ; the glands in the neighbourhood, superficial and deep, are also indurated and infiltrated in the same way. On dissection, it is found that the vessels for some distance around are firmly plugged with coagulated blood ; and this, therefore, is the reason that in this disease severe hæmorrhage is never seen. The nerves, in one instance, in which they were dissected by Barthez and Rilliet, were found to have the same appearance as the other tissues in the middle of the gangrene ; but this was found not to have extended further than the neurilemma ; the white substance of Schwann having quite a normal appearance.

With the exception of the lungs, other organs do not seem to be affected by this disease. In the lungs small nodules of pneumonia are invariably found, due either to the general blood condition or, as I should think more probably, to the conveyance by the blood of small masses of putrid slough from the gangrenous ulcer to the lung.

ETIOLOGY.—Gangrene of the mouth, like all the diseases previously described, is predisposed to by all conditions tending to impair the general health of the patient ; yet it is rare, and only in very weakly, badly nourished, and scrofulous children, that bad hygienic conditions are sufficient to produce this grave affection. In by far the majority of cases it follows some acute illness by which the health of the child has been greatly undermined. The disease of all others which seems more especially to predispose to this affection is measles. Of ninety-eight cases collected by M. Tourdis, in forty-one, or nearly half the cases, it followed measles. In nine it followed intermittent fever ; in nine, typhoid ; in seven it is put down as due to calomel ; in six it followed pertussis ; in five, scarlet fever ; in five, enteritis ; and the remaining cases followed various diseases.

Again, of forty-six cases collected by MM. Boulez and Caillault, in thirty-eight it followed attacks of measles.

It is most common between the ages of two and five, but may occur at any age between one and twelve years.

TREATMENT is both local and constitutional ; but the local is much the more important, and must be carried out thoroughly and energetically from the first moment that the disease has been discovered.

It consists in the application to the ulcer of strong hydrochloric or

nitric acid : the latter is most frequently used. A small piece of lint should be tied round the end of a stick of convenient size, and this dipped in the acid should be applied most thoroughly to all the ulcerated part. Care should be taken not to touch more than possible the healthy tissues around, and the tongue can be kept out of the way with a spatula, but every part of the ulcer must be thoroughly mopped out. And this must be done directly the disease is discovered, for the ulcer advances so rapidly that the delay of a day, or even of a few hours, seriously lessens the little patient's chances of recovery. The mouth should then be thoroughly washed out with water mixed with Condy's fluid, or solution of chlorine or carbolic acid ; and a piece of lint soaked in the solution used should be kept constantly applied to the ulcerated surface : this dressing should be changed at least every four hours, and at every change the parts should be most thoroughly washed.

One thorough application of the strong acid may be sufficient. If the ulcer continues to look dirty when examined the next day, and shows any signs of spreading, it should be again mopped out. If this application be made early in the disease, the patient has some chance of recovery, but otherwise the case is almost hopeless. A linseed meal poultice should be applied to the outside of the cheek.

The constitutional treatment consists in supporting the little patient with tonics, good nourishment, and stimulants. Chlorate of potass may be given in doses of five to ten grains every four hours. Dr. Burrows has treated cases of this disease successfully by this remedy alone, without any other local application than a chloride of soda gargle ; yet I should be sorry to trust to this medicine alone, even in very mild cases, and should not hesitate for a moment in any case about applying the strong acid. The secretions should be attended to ; and if purgatives are required, castor oil should be given. Mercury in any form should not be administered.

During the whole progress of the disease the ulcer must be daily examined ; one must not be satisfied with the general appearance of the patient, as this is often most deceptive. Whatever prognosis be given, it should be regulated much more by the state of the ulcer than by the general symptoms.

7. Glossitis—Simple Inflammation of the Tongue.

The various inflammations of the mucous membrane of the tongue have been described in the preceding sections with those of the mucous membrane of the mouth.

The disease now to be spoken of is an inflammation of the substance of the tongue, a very rare disease, sometimes caused by direct injury, by contact with irritative or corrosive substances, by scalding drinks, and by the bites or stings of insects.

Occasionally, it arises in the course of, or during the convalescence from, some one of the exanthematous fevers. I have known it

to occur, without apparent cause, after an attack of typhus fever, in a boy aged 17, seemingly strong and healthy.

Wood says it may be produced by a direct propagation of inflammation from the tonsils.

It is, however, far more frequently due to the action of such medicines as mercury than to any other cause, and these cases will be further spoken of in treating of salivation.

The symptoms of the disease are swelling, tenderness, and increased redness in the inflamed organ ; the whole tongue becomes enlarged. Generally commencing at the tip, which becomes bright red in colour, tense, shining, and painful, it in a few hours may extend backwards throughout the whole substance of the tongue, and cause it to enlarge so much as to fill the whole mouth, and even to protrude from it.

The floor of the mouth may be pushed down, the soft palate elevated, and the epiglottis so much pressed upon as seriously to impede respiration, and render suffocation possible. In such severe cases as these, the power of articulation is completely lost, and deglutition is rendered exceedingly difficult.

The breath becomes very fœtid, and altogether the patient is reduced to the most distressing condition. There is always some constitutional disturbance; the pulse is quick, hard, and bounding ; the temperature is raised ; there is a complete loss of appetite, and much thirst. The inflammation may extend to the surrounding parts ; the sublingual and submaxillary glands can be felt enlarged, hard, and tender under the jaw.

Under treatment the inflammation invariably terminates in resolulution, but if neglected suppuration or gangrene may occur. After an attack of Glossitis the tongue may remain permanently enlarged, so as to protrude beyond the teeth, requiring, other means failing, the removal of a portion of it.

Treatment is very simple and efficient. Free incisions should be at once made into the inflamed part on each side of the raphé, so as to allow the free escape of blood and serum, and in a few hours this will be found to have reduced the swelling immensely.

Saline purgatives should then be administered, and the patient, if otherwise well, should be kept on a low and farinaceous diet for a few days ; and this treatment, followed by a course of tonics, will be all that is required for the patient's thorough restoration to health.

If, however, permanent enlargement of the tongue results from one of these attacks, before resorting to operation, Dr. Druitt recommends that gentle pressure should be applied to the enlarged and protruded part. In a case of the kind under his notice, he found that the continuance of the protrusion was owing, first to the impaired function of the recently-inflamed organ, and secondly to some amount of constriction by the teeth. In this case, the application of gentle pressure was completely successful.

8. Ulceration of the Tongue.

Of the various forms of ulcer, those due to aphthæ have been noticed under the head of aphthous stomatitis.

A second set of ulcers are due to the irritation of decayed teeth in a constitution otherwise impaired. They occur generally at the sides of the tongue, opposite the molar or canine teeth. They are not at all indurated, and the edges are generally pallid and free from all signs of inflammation. They are easily got rid of by the abstraction of the offending tooth, and the use of astringent applications, nitrate of silver, sulphate of copper, &c., and a course of tonic treatment.

A third set of ulcers are due to syphilis. They are to be recognised by being elongated and irregular in shape, with glazed surface and indurated base, and by having swollen and indurated margins protruding above the level of the ulcer. They are not so tender, as a rule, as other ulcers, and they are sometimes very difficult to cure. Their diagnosis is, of course, helped by the history of the case, and the presence of the signs of syphilis on other parts of the body.

It is important to be able to diagnose these syphilitic ulcers from cases of cancer; it is sometimes exceedingly difficult, and can only be cleared up by time. The syphilitic ulcers have the character above described, and do not spread rapidly, whereas in cancer the ulcer is generally circular in shape, has eroded edges, and spreads very rapidly.

Syphilis may be also present in the tongue in the form of syphilitic tubercles. These are generally found deeply situated in the substance of the tongue, and are round, hard, indurated, and irregularly circumscribed masses. The surface of the tongue above them is redder than natural, and has more or less of a coppery colour.

The treatment consists in the administration of small doses of bichloride of mercury; either alone, or with iodide of potassium in the decoction of sarsaparilla, or some other convenient vehicle. They are very difficult to get rid of, and the treatment must be continued patiently for a long time. Local applications do not seem to have much effect.

Another condition of the tongue, which is sometimes due to syphilis, —but which, judging from some cases I have lately seen, is certainly not always due to that poison,—is that described as a psoriasis-like or eczematous condition of the tongue, when the tongue becomes glazed, smooth, and fissured in places, not at all indurated, and resembling more than anything else a piece of raw beef. In these cases, there has generally been more or less derangement of the digestive organs, but not sufficient to interfere seriously with the patient's health; in fact, with the exception of the discomfort produced by the condition of the tongue, the patients have been otherwise well.

Treatment in those due to syphilis is the same as for the syphilitic ulcer. In the other cases I have tried almost everything, and am loth to confess that they were, when discontinuing from a three or four months' attendance at the hospital, much in the same condition as

when they came. Nothing seemed to do much good, yet I found that a general tonic treatment, with a careful attention to the healthy performance of all the natural functions, and the restriction of the diet to the most easily digestible food, gave the greatest relief.

9. SALIVATION—PTYALISM.

An increase in the secretion of the saliva sometimes occurs to such an extent as to flow from the mouth, or necessitate its frequent discharge by spitting.

Excluding that produced by the action of medicines, the most common cause of an inordinate increase in this secretion is inflammation of the mucous membrane of the mouth, and consequent irritation of the salivary glands. Thus it is seen to be common in ulcerative stomatitis, cancrum oris, &c. Various gastric disorders have the same effect, as nausea, &c.

Pyrosis has by some authorities been considered to consist of nothing more than saliva swallowed,—an opinion which has received some support from the observation of Frerichs, that it sometimes contains a considerable quantity of sulpho-cyanide of potassium; but Dr. Wilson Fox thinks that this admixture may be due to some saliva swallowed, or mixed with the fluid during its ejection.[1]

Any disorder of the throat, as tonsillitis, also causes great increase of saliva.

It sometimes accompanies pregnancy, and in certain diseases, as hysteria, hydrophobia, and some states of mania, it is not uncommon.

Generally, salivation subsides on the removal of the cause by which it originated; if not, astringent washes may be used.

The salivation produced by medicines differs much from the idiopathic disease.

Mercury is the medicine by which it is most frequently produced; but various preparations of gold, copper, lead, arsenic, antimony, and bismuth, sulphuric acid, iodine, and iodide of potassium, with castor oil, foxglove, and prussic acid, have also been the cause of it in some people.[2]

Among the first symptoms of mercurial salivation is noticed a coppery taste in the mouth, soon followed by swelling and tenderness of the gums, and increase in the salivary secretion. The breath soon becomes very fœtid. As it progresses the teeth become quite loose, the gums very much swollen and spongy, and the tongue large and flabby, covered with thick white fur, with at the edges indentations corresponding to the teeth. Extensive ulceration may set in on the gums, spread over the whole mucous membrane of the mouth, and sometimes take on a gangrenous character. The face and neck are also more or less swollen, and the glands in the neighbourhood become inflamed, tense, and very tender.

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[1] Reynolds' System of Medicine, vol. ii. p. 797.　　[2] Guy's Forensic Medicine.

With this severe local inflammation there is always more or less heat of skin, frequent pulse, much thirst, and entire loss of appetite. The gums and teeth, in the early stages, become so tender, that it is impossible for the patient to take any solid food.

The duration of this condition is very variable; it may not last for more than a few days in some people, whilst in others it lasts for some considerable time after the mercury has been stopped.

In very severe cases the whole mouth, with its appendages, is so much swollen that it can hardly be opened; the patient is utterly unable to articulate, and deglutition is scarcely possible. The inflammation may extend to the throat, and there is generally intense pain in the gums and teeth.

The ulceration about the teeth may be so extensive as to render them so loose that they easily fall out, and even portions of the maxillary bones may be denuded and exfoliate.

TREATMENT.—Mild cases of mercurial salivation require no treatment; they get well immediately on leaving off the mercury.

Severe cases, however, sometimes require much care and attention. If there is much pain, opium should be given to relieve it, and chlorate of potass in doses of ten grains in decoction of cinchona should be given every four hours. The mouth should be frequently washed out with warm non-irritating washes at first, and then, as soon as the inflammation begins to subside, with slightly astringent liquids. The ulcerations, when present, may be treated as in ulcerative stomatitis, with alum, nitrate of silver, &c.

Fœtor of breath may be corrected by the use of solutions containing free chlorine, Condy's fluid, carbolic acid, or creosote.

A solution of acetate of lead—two or three grains to the ounce—has been much recommended in these cases, and at first it may be combined with a little opium to allay pain.

10. MALADIES OF DENTITION.

Before speaking of these diseases, it will not, perhaps, be out of place here to notice first a few facts with regard to the natural process of dentition.

The teeth are developed in two sets; the first set called the milk teeth: the temporary or deciduous set are smaller and less numerous, to suit the size of the jaw, than the second or permanent set.

The milk teeth are twenty in number—four incisors, two canines, and four molars in each jaw.

Mr. Marshall gives the following formula of them :—

$$\frac{M_2 \quad C_1 \quad I_4 \quad C_1 \quad M}{M_2 \quad C_1 \quad I_4 \quad C_1 \quad M_2}.$$

They make their appearance in the following order :—

The central incisors between the 5th and 7th months.
	lateral	,,	,,	,,	6th	,,	9th	,,
,,	first molars	,,	,,	9th	,,	15th	,,
,,	canines	,,	,,	15th	,,	18th	,,
,,	second molars	,,	,,	18th	,,	24th	,,

The lower central incisor is generally the first to appear.

Cases, though, have been observed where, at the time of birth, one or more teeth have been present, and others in which no teeth have appeared for more than two years.

Delayed dentition is, however, always due to rickets, whilst precocious dentition has been said to be due to a tubercular disposition, but this is rather doubtful.

The permanent set of teeth, thirty-two in number, consist of four incisors, two canines, four bicuspids, and six molars in each jaw. Formula according to Mr. Marshall :—

$$\frac{M_3 \quad B_2 \quad C_1 \quad I_4 \quad C \quad B \quad M_3}{M_3 \quad B_2 \quad C_1 \quad I_4 \quad C \quad B \quad M_3}.$$

They make their appearance in the following order, the first being the anterior molars :—

Four anterior molars at the 7th year.
,,	central incisors	,,	8th	,,
,,	lateral ,,	,,	9th	,,
,,	anterior bicuspids	,,	10th	,,
,,	posterior ,,	,,	11th	,,
,,	canines	,,	12th	to 12½	years.
,,	second molars	,,	12½	,, 14th	,,
,,	third ,,	,,	18th	,, 25th	,,

First dentition :—

The process of dentition varies considerably in different children. It is almost always accompanied by more or less pain and discomfort ; yet sometimes they are so slight as not to draw attention to the process, and it is not till the tooth is seen to be through, by examining the gum, that dentition is known to be going on.

At other times it is accompanied with great pain, swelling, and tenderness of the gums. They look tense and shining, and the position of the tooth is marked some time before its irruption by a distinct prominence on the edge of the gum.

The mouth is hot; the whole mucous membrane may become red; there may be a great increase in the secretion of the saliva, with a tendency to aphthous ulcerations about the corners of the lips and on the tongue. With this local inflammation there is always more or less febrile disturbance. The child is very feverish and irritable, frequently crying out as if in much pain, and passing very restless and sleepless nights. In these cases it is not unfrequent for small unhealthy ulcerations, with a sloughy appearance, to form on the summit of the gum, or around any tooth which has partially passed through it. (Odontitis).

All degrees may be noticed between this latter state and the normal, almost painless, process.

The various diseases which may complicate dentition are diarrhœa, cutaneous eruptions, cough, vomiting, various forms of inflammation of the mucous membrane of the mouth, and different affections of the nervous system, especially convulsions, essential paralysis, and strabismus.

It is, however, very essential to remember that although any one of these diseases may exist as a complication of dentition, yet such disease may exist at this time and be entirely independent of it; and sometimes the difficulty of distinguishing its true cause is very great, and cannot be at once determined.

The chief distinguishing symptom is the disappearance of the complication as soon as the evolution of the tooth is completed. Barthez and Rilliet also assert that when these diseases exist as complications of dentition, the constitutional disturbance produced by them is not so severe as when they are idiopathic. The symptom is the whole of the disease; the cough is not accompanied by râle; the diarrhœa is slight, and shows no other symptom of enteritis; and the vomiting, although very frequent during twenty-four or forty-eight hours, does not produce that grave condition observed when it is due to disease of the stomach.

Of the cutaneous eruptions, eczematous sores about the angles of the mouth and nose and behind the ears are very common, and some advise that these should be allowed to remain. The late Dr. Parrish, of Philadelphia, used to insist upon the importance, not only of not interfering with this salutary process of nature, but even of imitating it in cases of much obstinacy and danger, by keeping blisters open in the same situation.

Pruriginous strophulus is also very common during dentition.

There is no doubt that during the period at which dentition takes place all other diseases assume a much more serious aspect, and the danger depends, according to Guersant, on cerebral complications being much more frequent at this time than at others. Yet that the local irritation of teething seems to have nothing to do with this increased severity is rendered likely by it having been observed in many fatal cases of pneumonia, meningitis, &c., that the irruption of the teeth has taken place quite easily in the course of the illness.

It seems to be more probable that the increased danger of all diseases occurring at this period is due rather to the active general development of the organism that is going on than to the irritation of dentition, which is only one of its local expressions.

With regard to the proper management of children during this period, it is absolutely necessary to recognise all that has been previously said about diseases occurring at this time not being necessarily connected with, or dependent on, the process of dentition.

Formerly, by some, almost all the diseases of this period were considered due to the local irritation produced by teething, and the gum-lancet was used much more frequently than was necessary.

It is rarely indeed that the gums require lancing. It may be done with good results when the tooth is nearly through, when in at least a day or two it would rise through the gum; then some pain and irritation to the child may be relieved by cutting through the thin gum, and at once setting free the tooth: or if the gums be much swollen, shining, tense, and red, they may be scarified, to allow the escape of some blood and to relieve the inflammation: but in these cases it should always be explained to the mother why it is done; otherwise, the tooth not appearing soon afterwards, the mother will be disappointed, and think that the lancing has been improperly done. It may be necessary to do this two or three times before the tooth may appear.

Again, in cases of constitutional disturbance, as diarrhœa, vomiting, convulsions, &c., if the irruption of the teeth be actively going on, and the gums are much inflamed, lancing them will frequently do good.

If there be any fever, some simple febrifuge medicines may be given. Dr. West recommends bicarbonate of potass with citric acid; and if the child be very restless and irritable, two or three drops of the tincture of hyoscyamus may be added to each dose.

The diet should be most carefully regulated, and it should be seen that the nourishment be not given too often. The heat and dryness of the mouth so often present cause the child to cry continually for the breast, for the sake of the soothing sensation produced by the milk; in this way the child is very apt to overload its stomach, and produce colicky pains, diarrhœa, and vomiting.

Tepid baths are also of great service in allaying irritation and general febrile disturbance.

If there are any small aphthous ulcers present, they must be treated as before described in Aphthous Stomatitis. Generally in these cases there is more or less disturbance of the digestive organs.

In that severe form of ulceration to which the name of Odontitis has been given, small doses of chlorate of potass have been found to have the most beneficial effect. Local depletion by leeches is also extremely useful, and one or two may be applied with very good effect behind the angles of the jaw.

In these cases the submaxillary glands frequently become much swollen and tender.

In the second dentition the local symptoms are of the chief importance. These are various neuralgiæ of the teeth and gums, caries of the teeth, inflammatory toothache, and the like, which are met with most frequently in weakly, strumous children. For such maladies, change of air, agreeable exercise, good living, with tonic treatment, may be all that is required.

But during the cutting of the second set of teeth, and also during the development of the " wisdom teeth," various anomalous conditions of the digestive and nervous systems are often observed,—such as irritative dyspepsia, chorea, and convulsions,—which require the appropriate forms of treatment described elsewhere in this System of Medicine.

§ II.—Diseases of the Digestive System (*continued*).

C. Diseases of the Fauces, Pharynx, and Œsophagus.

1. Hæmorrhage from the Pharynx.
2. Relaxed Sore Throat.
3. Common Inflammation of the Fauces.
4. Gangrenous Inflammation.
5. Follicular Inflammation.
6. Simple Tonsillitis.
7. Herpetic Tonsillitis.
8. Chronic Tonsillitis.
9. Tumours of the Pharynx.
10. Inflammation of the Œsophagus.
11. Perforating Ulcer.
12. Paralysis.
13. Dilatation.
14. Stricture.

DISEASES OF THE FAUCES, PHARYNX, AND ŒSOPHAGUS.

By Charles E. Squarey, M.B. Lond.

1. Hæmorrhage from the Pharynx.

This disease has been included in the article on Hæmorrhage from the Mouth, and to those pages the reader is referred for its diagnosis and treatment.

Though rarely occurring in sufficient quantity to run from the mouth in a stream, slight hæmorrhage, just sufficient to streak the saliva with blood, is not by any means unfrequent, and from its almost invariably being thought to come from the lungs on account of the hawking and coughing necessary for its discharge, it causes much anxiety to the patient and his friends.

The condition of the throat and the absence of any chest symptom render the diagnosis very easy in the majority of cases, and the use of astringent gargles, with the administration of tonics, will be all that is required to arrest the bleeding.

2. Relaxed Sore Throat. Catarrhal Relaxation of the Throat.

This disease, which is marked by uneasiness or soreness in the throat, attended with slightly increased vascularity, and with swelling and œdema of the parts affected, is rarely attended with any constitutional disturbance.

It is not an uncommon affection in people of feeble constitution, and in those leading irregular and intemperate lives.

More or less uneasiness in the throat, increased by swallowing, with a dry, hacking cough, are the symptoms complained of. In some cases there may be increased secretion with continual expectoration.

On examination, the throat is seen to be more or less relaxed and swollen, the swelling having a humid or watery appearance. The uvula is much elongated and œdematous, and, by hanging down on the back of the tongue and epiglottis, it causes the dry tickling sensation and consequent cough so much complained of in this disease.

The larynx always participates in the general condition of the throat; the voice is altered in character, sometimes entirely lost, but

more frequently only much weaker and hoarser than natural. Much talking invariably aggravates this symptom.

Very little increase in the vascularity of the parts is observed. The attack is rarely accompanied with fever; the pulse remains quite normal; the tongue is moist, flabby, and furred, and generally indented at the edges by the teeth; the bowels are somewhat constipated.

There is not any thirst, and the appetite continues much the same as usual.

In some cases there may be a little lassitude and headache, but in the majority of cases the throat affection is all that is complained of; the patients being otherwise in their usual health and able to follow their ordinary occupation.

The attack generally comes on quite suddenly; it is most frequent in spring and autumn, especially in damp weather. It is most commonly produced by exposure to wet and cold; it may be secondary to an acute attack of inflammation of the throat, or it may depend on some derangement of the digestive organs.

Having once occurred, it is very liable to occur again.

The treatment in these cases is very simple. Tonics, as quinine, iron, &c., should be given to improve the general health of the patient; and the local irritation and relaxation should be combated by the use of astringent gargles.

By the use of the hand-ball atomizer, astringents may be very effectually applied to all parts of the throat. Tannin in solutions containing from 2 grs. to 10 grs. to the fluid ounce; tincture of per-chloride of iron, 5 to 20℔ to the f. ℥; alum, 1 to 10 grs. to the f. ℥; and nitrate of silver, ½ to 5 grs. to the f. ℥, will be found very useful.

The glycerine of tannin also would in such cases as these be most useful in bracing up the parts. It should be applied daily with a camel's-hair brush.

The uvula, if it causes much irritation and if it be not reduced in size by the astringents used, may be snipped off.

The diet should be good, consisting of easily digestible substances; and mild purgatives, such as the seidlitz powder, should be given when necessary.

3. COMMON INFLAMMATION OF THE FAUCES. SORE THROAT. ANGINA SIMPLEX.

A simple inflammation of the mucous membrane of the pharynx brought on by exposure to cold and damp. Some people are much more liable to it than others. Having once occurred, it is very liable to occur again.

The first symptoms are a sense of heat and dryness in the throat, with acute pain on swallowing, and more or less hoarseness of the voice. There is frequently a constant desire to cough without the patient being able to hawk up anything.

On examination, the fauces and pharynx generally are seen to be more or less inflamed, one side frequently more so than the other: the parts are swollen, and of a bright red colour; or, in severe cases, of a darker, almost livid red hue, with a dry, glistening, velvety appearance. The uvula and tonsils participate in the general inflammation, and the former, much elongated and œdematous, is frequently the cause of the constant cough. Patches of coagulated lymph may be seen forming a false membrane over various parts of the soft palate and pharynx, and the tonsils are not unfrequently covered with little white spots, the increased secretion of the glands. The inflammation commonly extends to the Eustachian tube and causes partial deafness; and it may spread downwards into the larynx and trachea, causing hoarseness, loss of voice, the peculiar laryngeal cough, and, in severe cases, seriously impeding the respiration.

A viscid mucus is after a time secreted and clings to the posterior part of the pharynx, causing a constant desire to expectorate. It sometimes very closely simulates the false membrane of diphtheria, especially in those cases where it is so viscid that coughing will not remove it.

Its position at the back of the pharynx, its smooth and regular edges not surrounded by a bright red margin of inflammation, will in most cases render the diagnosis easy: if not, its ready removal by the finger, without causing excoriation or bleeding, at once decides the case.

More or less injection of the skin is always present, and in some cases it is so bright as to resemble somewhat the rash of scarlet fever: but in these cases the injection is always limited to the face and neck, the upper part of the chest, and to the hands and forearms; it does not spread from these parts over the rest of the body, and it consists of a simple blush; it has not any of those fine red punctæ which are characteristic of the rash of scarlet fever.

' The disease is always accompanied with some constitutional disturbance; in many cases its invasion is marked by slight rigors, headache, and aching pains in the limbs. The tongue is broad and flabby, with a thick, yellowish, creamy fur on the dorsum, the breath always more or less fœtid, and the bowels generally constipated. There is much thirst, and entire loss of appetite. The temperature in many cases that I have taken has always been between 101° and 102° Fahr., the pulse varying from 100 to 120 beats in the minute.

The heat and dryness of the throat cause much discomfort, which on all attempts to swallow is intensely aggravated, the pain in some cases being very severe. Drinks not unfrequently regurgitate through the nostrils, and there is much pain and stiffness about the angles of the jaws. All the symptoms are much aggravated at night, and towards morning, on account of the mouth having become dry during sleep.

DURATION.—In ordinary cases the inflammation runs on for about a week, and then gradually declines, terminating almost always in

resolution. In weakly subjects suppuration may take place in the soft palate or in the back of the pharynx, or even superficial gangrene of the inflamed parts may occur; but both are very rare: the latter especially, as it is much more common in these patients for the throat to get into the relaxed condition previously described.

The presence of pus is easily diagnosed by the swelling and by the fluctuation felt on passing the finger down the throat—it should be immediately let out by a free incision.

The prognosis in all cases is good, though sometimes the cure is tedious. The only danger is the extension of the inflammation to the larynx.

CAUSES.—The most frequent cause of this complaint is exposure to damp and cold.

All causes tending to lower the general health predispose to this affection: overwork, especially when combined with the effects of a vitiated atmosphere, as is so often found in hospitals, gives rise so constantly on the least chill to this affection, that the name "hospital sore throat" is very commonly applied to it. It is chiefly met with during puberty and adult age, but may occur at all ages. One attack predisposes to others.

TREATMENT.—In all cases the treatment should be commenced by a dose of calomel at night, followed the next morning by the common black draught or some saline aperient; and this, with the frequent use of steam inhalations and the external application of hot fomentations, will frequently cut short the attack. If there is much swelling and pain, and the attack be accompanied with much fever in an otherwise strong and healthy constitution, two or three leeches may be applied with much benefit behind the angles of the jaws.

After the bowels have been freely opened, chlorate of potass in 10-grain doses, with a little dilute hydrochloric acid (mx. to $m$xv.) in tonic syrup of orange-peel and water, should be given every four hours. The diet should at this time consist of strong beef-tea, milk, and eggs; no stimulants are necessary, unless the patient be weak and low; he should be kept in a uniform temperature and as quiet as possible. Barley-water, linseed tea, or any other mucilaginous drink, may be taken *ad libitum*, and they frequently give great relief when there is much dryness and heat of throat.

If suppuration takes place, directly it is detected a free incision should be made to allow its escape. In making the incision the knife should be directed slightly towards the median line, and it should be made at the lowest point, so that there may be no bagging of pus below the opening.

After the inflammation has subsided quinine should be given, the diet improved, and port wine, when obtainable, taken two or three times a day. If the throat seems inclined to get into a chronic relaxed condition, astringent gargles or inhalations should be used

The inflammation almost invariably extends into the larynx, causing huskiness and pain, which are much aggravated by talking, and sometimes followed by aphonia.

Although causing much pain and discomfort, this disease is only in very severe cases accompanied with any constitutional disturbance. Ordinarily the pulse remains quite normal, the tongue a little furred, and the appetite only slightly impaired, though for two or three days solid food cannot be taken on account of the pain on swallowing.

The disease generally lasts for a week or so, in some cases much longer, and it may pass into a persistent chronic state, especially in weakly subjects, and when the weather is unfavourable.

It is brought on by exposure to cold and wet; and, as in the previous disease, some people are much more liable to it than others, the slightest changes in the weather being sufficient to produce it in them. Derangements of the stomach, the use of spices and hot drinks, have been mentioned amongst other causes.

All conditions tending to impair the general health predispose to this affection. It is chiefly met with in adults; it is rare in the young and at the later periods of life. One attack predisposes to others.

The treatment in these cases may be beneficially commenced by a dose of calomel, followed in two or three hours or the next morning by a saline aperient. And after the bowels have been effectually opened, tonics, such as quinine and iron, may be given three or four times a day; or the chlorate of potass—in 10-grain doses, with a little dilute nitric acid—in decoction of cinchona bark.

The ulcers when present should be touched with the solid stick of nitrate of silver; and if secreting freely and causing much fœtor of breath, solutions of carbolic acid—♏ xv. of concentrated acid to the ounce of water—or of chloride of lime applied by the hand-ball atomiser, or inhalations of creosote, will rapidly give great relief; other astringents, as alum, sulphate of copper, or acetate of lead, may also be used, either as gargles or in the form of the medicated spray.

The diet should consist of sops and mucilaginous drinks whilst there is much pain; the latter, by moistening the parts frequently, give great relief.

Various other ulcers are met with in the throat: they occur in the course of different diseases; as scarlet fever, typhoid fever, small-pox, or syphilis, and they sometimes are due to the action of mercury.

Those produced by syphilis are the only ones which are liable to be confounded with the ulcerated sore throat just described. They can, as a rule, be easily diagnosed by the following characters:—

Syphilitic ulcers are sharply defined, and are either circular in shape or serpiginous; their edges are swollen and indurated, and frequently have a distinct coppery tint; while the floor of the ulcer is grey. They cause little if any pain, and do not yield to ordinary treatment. The history and the presence of the symptoms of syphilis in other

parts of the body will also aid the diagnosis. For further particulars and for the distinctive characters of the other ulcers and their treatment, the reader is referred to the articles especially devoted to those subjects.

6. Tonsillitis.

Acute Inflammation of the Tonsils, Cynanche Tonsillaris, or Quinsy, is an inflammation of the substance of the tonsils, either one or both, and is accompanied with much constitutional disturbance.

The invasion of this disease in all but very mild cases is marked by a general feeling of malaise, by headache and aching pains in the limbs, with a sense of chilliness, or even distinct rigors; and at the same time there is felt some dryness or uneasiness in the throat, with acute pain shooting towards the ears on swallowing. The local and constitutional symptoms begin at the same time.

The uneasiness in the throat increases until it amounts to severe pain; swallowing becomes very difficult, and so painful as to cause a convulsive contraction of the features on each attempt; the pain shooting towards the ears. There is much tenderness behind the angles of the jaws, and they become so stiff that the patient with difficulty opens' his mouth sufficiently for a thorough examination to be made.

The glands of the neck become enlarged and hardened, and not unfrequently the neck itself is somewhat swollen and stiff.

As the disease advances, swallowing becomes almost impossible; fluids regurgitate through the nose, the mouth feels choked up from an increased secretion of tenacious saliva, the voice is much weakened and sometimes acquires a nasal twang; and in bad cases it may be entirely suppressed. There is more or less deafness, and if both tonsils are very much swollen there may be a sense of suffocation on lying down owing to the fauces being blocked up by the swollen glands; this more frequently occurs in children than in adults.

On examination in the early stages of the disease, the fauces are seen to be redder than natural, with the tonsils projecting slightly between the palatine arches. They gradually increase in size, and if both are affected they meet in the middle line. On their surface are seen, here and there, little white or yellowish spots, the increased secretion of the glands; these, when numerous and close together, form patches not unlike the false membrane of diphtheria. The uvula and soft palate always participate in the general affection, and the former, much swollen and elongated, is generally seen sticking to one of the tonsils.

The fever and general prostration that accompany these attacks are always very considerable. In many cases in which I have taken the temperature it has always been over 102° Fahr. In one case it was 104° Fahr. on the fourth day of the attack. The skin is moist and more or less injected, as in the Angina Simplex previously

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described. The pulse ranges between 100 and 120. The tongue is covered with a thick yellowish creamy fur; the bowels are usually much constipated, and the breath is intensely fœtid. There is generally much restlessness, especially at night time, and in severe cases there may be a little delirium.

These attacks vary greatly in severity. In mild cases, and in those coming under treatment in the early stages of the disease, the inflammation may terminate in resolution, but in most cases suppuration takes place. The presence of matter is indicated by the soft elastic feeling of the tumour, and generally distinct fluctuation can be felt. If no incision is made in it, a day or so before the matter escapes, a pale yellowish spot will be seen on the surface, indicating the point at which the matter tends to escape. Up to this time the patient suffers intense pain, but immediately on escape of the matter great relief is obtained. It generally occurs during some effort made by the patient in coughing, swallowing, or clearing his throat; it may, however, occur during sleep, without the patient being conscious of the time when it took place.

After the opening of the abscess recovery is generally very rapid, though for some time afterwards the tonsils may remain swollen and indurated; this is especially likely to happen in weakly, scrofulous patients, or after repeated attacks of the disease.

Both tonsils may be affected, but it is generally only on one side that suppuration takes place. The disease almost invariably terminates favourably. It takes about five or six days to arrive at its height, sometimes longer, in one case under my notice lasting till the tenth day.

The urine during the acute stage has been, in the cases I have examined, much diminished in quantity, very high-coloured, and of high specific gravity. The urea, in one or two cases was a little above the normal quantity; the chlorides were almost entirely absent. No albumen was present at any time.

This disease is most common during youth and adult age. Some people are extremely liable to it. One attack predisposes to others, yet those who have suffered from it when young generally cease to do so when they become old.

Its most frequent cause is exposure to damp and cold, so that it is most common during the spring and commencement of the winter.

TREATMENT.—In all cases the treatment should be commenced by free purgation, such as may be obtained by a dose of calomel, followed by a saline aperient draught. After this has thoroughly acted, chlorate of potass may be given, as in the previous throat affections. Hot fomentations should be applied, and these should pass up from underneath the chin to the top of the head, not round the neck. They should be changed frequently, and put on as hot as the patient can bear them. Steam inhalations should at the same time be used every three or four hours, and these will be found to give great relief.

The diet should be as nourishing as possible, and at this time should consist of strong beef-tea, eggs, and milk; if the patient is very much prostrated, brandy or port wine may be given.

Immediately that any fluctuation can be felt, or even before, if there is much pain, a free incision should be made into the tonsil, the knife being directed slightly towards the median line; even if no matter escapes, the bleeding will give more or less relief.

If there is much restlessness at night, an opiate may be given, and the best form is the Dover's powder, 10 grains mixed up with mucilage and water.

After the acute symptoms have passed by, tonics, such as quinine and iron, may be freely given. The diet should be very good, and plenty of port wine taken; if the tonsils remain large, the tincture of the perchloride of iron should be given in doses of from ℥ xx to ℥ xxx every three or four hours, and the tonsils may at the same time be painted over with a solution of equal parts of the tincture of iron and glycerine. Painting the enlarged tonsils daily with a strong solution of nitrate of silver has also been recommended; but in the majority of cases, where they remain so large as to cause serious discomfort it will be necessary to excise them.

7. HERPETIC TONSILLITIS.

This is a vesicular disease attacking the tonsils and the inside of the mouth generally.

Herpetic eruption may occur on any part of the oral cavity. On the mucous membrane of the mouth it much resembles aphthæ, and gives rise to the same symptoms,—that is, more or less discomfort or pain on chewing hard substances, taking hot drinks, or eating highly-seasoned food; it gets well in a few days without treatment.

When occurring on the tonsils or pharynx, it in some cases gives rise to much constitutional disturbance.

The invasion of the disease is marked in these cases by rigors and general malaise, followed at the end of twenty-four or thirty hours by a sharp burning sensation in the throat, and intense pricking pains shooting towards the ears on swallowing.

Generally one side only is affected.

On examining the throat, the tonsils and pharynx are seen to be of a bright red colour, and swollen, the former projecting between the arches of the palate; and if seen sufficiently early in the disease, a distinct vesicular eruption will be observed.

Generally, when first seen, the vesicles have already ruptured, leaving the appearance of little white spots on the tonsil, due to the macerated condition of the epithelium; or if the epithelium has been washed away, there are to be observed bright red excoriations or ulcers.

According to Trousseau, these spots may be now covered with a soft yellowish pultaceous exudation, and if the eruption has been copious,

portions of this exudative matter may join together and form irregular shaped patches much resembling the false membrane of diphtheria.

From the pharynx the inflammation may extend into the larynx, and cause some trouble and anxiety. The exudation remains for a day or so, then falls off, and the parts return to their normal condition.

There is sometimes a considerable amount of pyrexia present, the pulse may rise to 120, or even higher, the tongue is more or less furred, the appetite entirely gone, there is some thirst and generally slight constipation.

The disease runs to its height in a few days, then as rapidly gets well. It is generally produced by exposure to damp and cold.

Herpetic Tonsillitis may be the only morbid condition existing, but more frequently it coexists with some other acute disease, as pneumonia, pleurisy, intermittent fever, and certain catarrhal maladies.

Its relation to these diseases is at present entirely unknown. Its presence is neither of good nor bad augury with regard to the probable issue of the disease which it accompanies.

Its DIAGNOSIS from diphtheria is in many cases extremely difficult, especially when it is not accompanied by any herpetic eruption on the face or elsewhere. The general condition of the patient will be the great guide in these cases, the constitutional symptoms being not nearly so severe in this affection as in diphtheria; yet it must be borne in mind that the latter disease is frequently very insidious in its origin.

In all cases of doubt, and especially when diphtheria is epidemic at the time, the patient should be treated for the graver malady.

The TREATMENT of the herpetic affection is very simple. The bowels should be relieved by mild purgatives, as the seidlitz powder, and iron or chlorate of potass should be given internally, as mentioned in the previous pages.

Slightly astringent gargles of alum or borax may also be used. Mucilaginous drinks and sops should be taken for the first day or so, and then the diet should be improved as the patient desires.

8. CHRONIC TONSILLITIS. HYPERTROPHY OF THE TONSILS.

A chronically indurated and enlarged condition of the tonsils is sometimes the result of repeated attacks of inflammation; but it occasionally arises independently of any inflammatory origin, in weakly, scrofulous children.

The enlarged tonsils cause sometimes much difficulty in swallowing and in breathing—the latter is always more or less noisy. They are also extremely liable to fresh attacks of inflammation on the least exposure to damp and cold, and may then become so large as to cause suffocation by completely blocking up the throat. In weakly, delicate children, if the respiration is much impeded, they may cause some deformity of the chest.

Under the influence of a course of tonic treatment, the swellings may subside as the child grows older ; but if not, and they are causing any distress, they should be excised.

9. TUMOURS OF THE PHARYNX.

Retro-pharyngeal Abscess is a term applied to denote a collection of pus in the cellular tissue posterior to the pharynx, and situated between it and the cervical vertebræ.

It is not a very common affection, and is more frequent in children than in adults, resulting from acute pharyngitis, from disease of the cervical vertebræ, or it may occur in the course of the acute specific fevers.

The Symptoms are, pain at the back of the pharynx, with difficulty of swallowing, and sometimes much oppression of the breathing. When high up, it can be seen forming a tumour at the back of the pharynx, and on touching it distinct fluctuation will be felt. When lower down, the diagnosis is not so easy ; but difficulty of swallowing, oppression of the breathing, with deep-seated pain in the pharynx coming on rather rapidly, are the symptoms which should lead one to suspect it. A bougie might be passed to find out where the obstruction lies.

It is always very serious—many fatal cases have been recorded, and one occurred under my notice at the London Fever Hospital.

The Treatment is to open the abscess as soon as possible ; the incision should be made in the middle line at the lowest part of the tumour, to favour the escape of pus and to prevent bagging. The patient will require much support. Stimulants should be freely given, and nutritive enemas or the stomach-pump used if necessary.

POLYPI OF THE PHARYNX.—These generally spring from the posterior nares, and hang down into the pharynx, blocking up and rendering difficult the act of swallowing, and sometimes causing oppression of the breathing. They may arise from the sides of the pharynx, but when having this seat they are generally malignant growths.

On examination, the disease is at once detected. The treatment is removal.

CANCER OF THE PHARYNX.—A cancer may arise from the walls of the pharynx—it is then generally epithelial ; but it is more frequently connected with the cervical vertebræ, and grows forward, forming a tumour, at last eating its way into the pharynx.

It is easily recognised by the peculiar characters of cancerous tumours, by the implication of the neighbouring glands, and by the cachectic condition of body which is its accompaniment.

The Prognosis is always most grave ; and the Treatment consists in the alleviation of pain by opium or other sedatives, and the careful support of the patient by food and stimulants.

10. ŒSOPHAGITIS. INFLAMMATION OF THE ŒSOPHAGUS.

This is a very rare disease, and usually arises from mechanical violence or from the direct application of irritating or corrosive substances.

The Symptoms are entirely local, consisting of pain along the course of the œsophagus, increased by swallowing, accompanied with expectoration of a thick viscid mucus, or flakes or tubes of false membrane sometimes tinged with blood. The pain may be referred to the epigastrium, or it may be between the shoulders; there is sometimes tenderness on deep pressure.

Very little is known concerning the simple inflammatory state; when due to mechanical violence,—as from the use of the stomach-pump,—soreness, with pain and some difficulty on swallowing, are felt for a few days, and then the patient is well. If due to acrid poisons, ulceration and stricture generally result.

The most frequent causes are direct application of some acrid, corrosive, or hot substance—taken either accidentally or suicidally—and mechanical violence from sharp rough foreign bodies introduced into the œsophagus, or from violence in the use of the stomach-pump.

It may be propagated downwards from the fauces or larynx, as in diphtheria and thrush. It may arise in the course of some of the acute specific fevers, as scarlet fever, small-pox, and typhoid fever, but the ulcer produced by this latter disease does not tend to contract afterwards. It may be secondary to syphilis. When occurring in the course of other diseases, it is rarely to be recognised during life.

At the post-mortem examination, the mucous membrane is seen to be of a deep red colour, and covered with more or less soft and granular lymph.

Cases have been recorded in which the œsophagus has been found completely lined with false membrane.

The difficulty in the diagnosis consists in distinguishing it from similar conditions of the stomach.

PROGNOSIS.—It generally terminates in resolution; but if it gets into a chronic state, it may cause thickening of the coats, and stricture.

The TREATMENT consists in relieving the symptoms by warm poultices, and the application of two or three leeches or blisters to the neck or epigastrium.

Opium should be given to relieve pain. A mild course of mercury should be tried if the disease becomes chronic. The bowels should be carefully attended to, and the diet should consist of easily swallowed and nourishing materials.

11. PERFORATING ULCER OF THE ŒSOPHAGUS.

In these cases, the symptoms are the same as those of simple inflammation of the œsophagus, but they are aggravated.

The pain is more severe and more localised, the difficulty in swallowing is much greater, and the act is sometimes impossible; the expectorated mucus is always more or less tinged with blood.

It is liable to cause spasm of the œsophagus, and it is very important to remember this, as most serious injury may be done in these cases by attempting to pass a bougie.

The PROGNOSIS is always very grave.

The TREATMENT consists in relieving the symptoms and attending carefully to the diet and the bowels.

12. PARALYSIS OF THE ŒSOPHAGUS.

This disease is very rare. It is met with chiefly in the course of other diseases; thus it is seen in the last stages of progressive muscular atrophy, in glosso-laryngeal paralysis, in the paralysis of the insane, and in other nervous affections. It is one of the sequelæ of diphtheria, and it may be simulated by hysteria.

When not complete, solids are generally swallowed better than liquids. If complete, the patient must be fed by nutritive enemas, or the stomach-pump.

PROGNOSIS is always most grave, except when it occurs in hysterical patients.

TREATMENT.—When due to hysteria, the general conditions must be attended to; in other cases, the effect of electricity, blisters, and nux vomica, may be tried.

13. DILATATION OF THE ŒSOPHAGUS AND PHARYNX.

This disease is generally associated with stricture of the œsophagus, but it may occur independently of narrowing.

The dilatation may be in the form of a pouch, made either by distension of all the coats at one particular spot, or by protrusion of the mucous membrane between the muscular fibres (Œsophagocele); or it may be a general distension of the tube in some part of its length.

The Symptoms in all cases are increasing dysphagia and regurgitation of the food taken, after a longer or shorter interval. There is always a sensation as if the food was arrested a little above the stomach, and this in some cases gives rise to painful and protracted efforts to swallow. Immediate relief follows vomiting. The dilated part, when full of food, can frequently be felt as a tumour on one side of the neck, and can be emptied by pressure.

It is a disease of the prime and decline of life. Males are more liable to it than females.

Little can be done in the way of treatment. Bougies should be passed every now and then, and in passing them the instrument should

10. ŒSOPHAGITIS. INFLAMMATION OF THE ŒSOPHAGUS.

This is a very rare disease, and usually arises from mechanical violence or from the direct application of irritating or corrosive substances.

The Symptoms are entirely local, consisting of pain along the course of the œsophagus, increased by swallowing, accompanied with expectoration of a thick viscid mucus, or flakes or tubes of false membrane sometimes tinged with blood. The pain may be referred to the epigastrium, or it may be between the shoulders; there is sometimes tenderness on deep pressure.

Very little is known concerning the simple inflammatory state; when due to mechanical violence,—as from the use of the stomach-pump,—soreness, with pain and some difficulty on swallowing, are felt for a few days, and then the patient is well. If due to acrid poisons, ulceration and stricture generally result.

The most frequent causes are direct application of some acrid, corrosive, or hot substance—taken either accidentally or suicidally—and mechanical violence from sharp rough foreign bodies introduced into the œsophagus, or from violence in the use of the stomach-pump.

It may be propagated downwards from the fauces or larynx, as in diphtheria and thrush. It may arise in the course of some of the acute specific fevers, as scarlet fever, small-pox, and typhoid fever, but the ulcer produced by this latter disease does not tend to contract afterwards. It may be secondary to syphilis. When occurring in the course of other diseases, it is rarely to be recognised during life.

At the post-mortem examination, the mucous membrane is seen to be of a deep red colour, and covered with more or less soft and granular lymph.

Cases have been recorded in which the œsophagus has been found completely lined with false membrane.

The difficulty in the diagnosis consists in distinguishing it from similar conditions of the stomach.

PROGNOSIS.—It generally terminates in resolution; but if it gets into a chronic state, it may cause thickening of the coats, and stricture.

The TREATMENT consists in relieving the symptoms by warm poultices, and the application of two or three leeches or blisters to the neck or epigastrium.

Opium should be given to relieve pain. A mild course of mercury should be tried if the disease becomes chronic. The bowels should be carefully attended to, and the diet should consist of easily swallowed and nourishing materials.

11. PERFORATING ULCER OF THE ŒSOPHAGUS.

In these cases, the symptoms are the same as those of simple inflammation of the œsophagus, but they are aggravated.

The pain is more severe and more localised, the difficulty in swallowing is much greater, and the act is sometimes impossible; the expectorated mucus is always more or less tinged with blood.

It is liable to cause spasm of the œsophagus, and it is very important to remember this, as most serious injury may be done in these cases by attempting to pass a bougie.

The PROGNOSIS is always very grave.

The TREATMENT consists in relieving the symptoms and attending carefully to the diet and the bowels.

12. PARALYSIS OF THE ŒSOPHAGUS.

This disease is very rare. It is met with chiefly in the course of other diseases; thus it is seen in the last stages of progressive muscular atrophy, in glosso-laryngeal paralysis, in the paralysis of the insane, and in other nervous affections. It is one of the sequelæ of diphtheria, and it may be simulated by hysteria.

When not complete, solids are generally swallowed better than liquids. If complete, the patient must be fed by nutritive enemas, or the stomach-pump.

PROGNOSIS is always most grave, except when it occurs in hysterical patients.

TREATMENT.—When due to hysteria, the general conditions must be attended to; in other cases, the effect of electricity, blisters, and nux vomica, may be tried.

13. DILATATION OF THE ŒSOPHAGUS AND PHARYNX.

This disease is generally associated with stricture of the œsophagus, but it may occur independently of narrowing.

The dilatation may be in the form of a pouch, made either by distension of all the coats at one particular spot, or by protrusion of the mucous membrane between the muscular fibres (Œsophagocele); or it may be a general distension of the tube in some part of its length.

The Symptoms in all cases are increasing dysphagia and regurgitation of the food taken, after a longer or shorter interval. There is always a sensation as if the food was arrested a little above the stomach, and this in some cases gives rise to painful and protracted efforts to swallow. Immediate relief follows vomiting. The dilated part, when full of food, can frequently be felt as a tumour on one side of the neck, and can be emptied by pressure.

It is a disease of the prime and decline of life. Males are more liable to it than females.

Little can be done in the way of treatment. Bougies should be passed every now and then, and in passing them the instrument should

be made to glide as nearly as possible along the posterior part of the larynx.

PROGNOSIS is always very grave, the patient sooner or later dying of starvation.

14. STRICTURE OF THE ŒSOPHAGUS.

Obstruction to the passage of food into the stomach may be due to spasmodic stricture of the œsophagus, to organic stricture, or to disease of neighbouring parts compressing the œsophagus.

SPASM OF THE ŒSOPHAGUS.—A morbid contraction of the muscles of the tube, occurring in highly nervous hysterical subjects, and causing more or less difficulty in swallowing.

The Symptoms are—sudden difficulty in swallowing, coming on at irregular intervals and persisting for a longer or shorter time.

On attempting to swallow, the food is either rejected at once, if the spasm is at the upper part of the tube, or, if lower down, it is retained for a longer time and then regurgitates. Occasionally, when the food has been in contact with the stricture for some time, the latter yields, and some food finds its way into the stomach. Solids are generally swallowed better than liquids; but, as a rule, any sort of food causes the spasm. The attacks are sometimes accompanied with great pain, and are followed by great lassitude and depression.

A sense of constriction is often felt even when an attempt to swallow is being made; and this feeling in the throat constitutes what is sometimes termed the Globus Hystericus. Occasionally the irritation extends to the larynx and lungs, and causes much embarrassment to the respiration, with a sense of impending suffocation.

The duration of the attacks is very various, some only lasting for a few hours, others continuing with more or less complete remissions for months or years.

From organic stricture—the only disease with which it can be confounded—it is at once distinguished by its sudden accession, and by the occasional remission of symptoms, allowing the patient to swallow quite easily. During a remission, a large bougie can be passed without difficulty; and even during the spasm, if the bougie be gently pressed onwards for some little time, the spasm will relax, and the instrument will pass into the stomach.

CAUSES.—In those predisposed to this affection, the least irritation of the mucous coat is sufficient to bring on an attack.

The predisposition consists in an excitable state of the nervous system, such as is met with in hysterical and hypochondriacal people, together with an anæmic and debilitated condition of the body. Emotions of various kinds, diseases existing elsewhere, as affections of the larynx, stomach, or uterus, or in bad cases even the mere attempt to swallow saliva, may be sufficient to excite the spasm.

At the same time, it must be remembered that it may be produced by inflammation, or the presence of an ulcer in the mucous coat; so that in all cases in passing instruments the greatest care should be used.

The PROGNOSIS is rarely grave, although the cure in some cases is exceedingly tedious and difficult.

TREATMENT.—When due to hysteria, thorough change of air, exercise, occupation for the mind, and pleasant companionship, should be recommended, together with the internal administration of tonics and antispasmodics.

Strychnia has been found very useful in some cases; and quinine in others where the spasm has been regularly intermittent.

Locally, warm poultices, the hypodermic injection of morphia, blisters, setons, &c. may be tried. In purely nervous affections the bougie should not be passed.

If due to inflammation or ulcer, the treatment must be that already indicated for those diseases.

ORGANIC STRICTURE OF THE ŒSOPHAGUS, and those diseases of neighbouring parts which by pressure cause dysphagia, come properly within the domain of the surgeon, so that here only a short notice will be taken of them.

ORGANIC STRICTURE.—The chief symptom of this complaint is gradually increasing dysphagia.

At first this difficulty may be only felt at times; then it is always felt, though at some times much worse than at others—this being due to spasm, occasioned by irritation of the diseased parts. Years may elapse before any great difficulty occurs. At first solids are passed with difficulty, then they cannot be passed at all, and regurgitate.

Dilatation of the œsophagus above the stricture takes place in some cases; so much so, that large quantities of food can be taken, and will remain down some time—the patient always having a sensation of its "sticking somewhere"—and at last, when regurgitated, the pieces of food are generally covered with more or less mucus, a little blood or pus. There is no acid reaction in the mucus coming from the œsophagus, and this fact is sometimes of service in the diagnosis from gastric disease. Some little quantity of food may pass the stricture and get into the stomach.

Arrived at this stage, emaciation commences, and proceeds very rapidly. There is rarely any great pain or hæmorrhage.

The irritation and inflammation may extend to the larynx, and cause much oppression of the breathing; or, by implicating the recurrent laryngeal nerve, may cause continual cough. At last the patient dies from inanition.

The most common seats of the stricture are the upper third of the œsophagus and its cardiac end. When situated high up, the œsophagus is diminished in size below.

This is the general course of symptoms in stricture produced by disease of the œsophagus itself, such as cancer of the œsophagus, and induration and contraction of the walls from the action of corrosive substances.

When the dysphagia is due to diseases existing external to the œsophagus, the constriction is rarely so complete.

The various conditions which will thus cause dysphagia are—polypi of the pharynx, retro-pharyngeal abscess, post-pharyngeal tumours, as cancer, springing from the bodies of the vertebræ; œdema at the back of the epiglottis, tumours in the neck, such as enlarged glands; carotid aneurism, or enlarged thyroid; aneurism of the innominate artery, or of the aorta; intrathoracic tumours, such as enlarged bronchial glands; cancer of the apex of the lung, distended pericardium, dislocation of the sternal end of clavicle backwards, and impaction of foreign bodies in the gullet.

When due to aneurism, a bougie should never be passed.

For the characteristic symptoms and treatment of these various diseases the reader is referred to books on Surgery.

§ II.—DISEASES OF THE DIGESTIVE SYSTEM (*continued*).

D. DISEASES OF THE INTESTINES.

1. ENTERALGIA.
2. ENTERITIS.
3. OBSTRUCTION OF BOWELS.
4. ULCERATION OF BOWELS.
5. CANCEROUS AND OTHER GROWTHS IN THE BOWELS.
6. DISEASES OF THE CÆCUM AND APPENDIX VERMIFORMIS.
7. COLIC.
8. COLITIS.
9. DYSENTERY.
10. DISEASES OF THE RECTUM AND ANUS.
11. INTESTINAL WORMS.

ENTERALGIA.

By John Richard Wardell, M.D. F.R.C.P.

Definition.—Enteralgia is a painful affection of the intestines, of neuralgic character, generally accompanied with constipation and flatus. It may come on gradually in a dull and obtuse manner, but in the great majority of instances its supervention is sudden, and the pain is sharp and violent. It is, correctly speaking, visceral neuralgia, and mostly occurs in neurotic individuals. The common accompaniments of inflammation are absent. The skin is cool, the pulse is not accelerated, and the heart's impulse is rather subdued than augmented. Its attacks are paroxysmal. It shifts its position in the abdomen. It is often a pain reflected by distal disease, but if continuous it may end in inflammation.

Synonyms.—Enteralgia, Tormina, Dolor Colicus, Colicodynia, Spasmus Intestinorum (various Authors). Ileus Spasmodicus (Sauvages); Spasmus Ventriculi (Wiessner). Some writers have confounded it with Gastrodynia, or Gastralgia. In the vernacular the affection is identical with Pain of the Intestines, Spasm of the Bowels and Belly-ache, Pain in the Belly, Gripes, and Cholick, or Cholick Colic.

Causes.—The causes of this complaint are to be regarded as those which are Predisposing or Remote, and those which are Proximate or Exciting.

Under the head of the first-named may be mentioned the influence of sex, and it is beyond dispute that females are more prone to this affection than males; their greater sensitiveness, and their susceptibility to moral emotions, favour the development of nervous diseases; and the sympathy of the uterus and its appendages, as familiarly known, in marked manner reacts upon the cerebro-spinal and ganglionic systems. The particular temperament of the patient will confer a proneness to, or tend to give an immunity from this complaint, those who are nervous and melancholic being more liable to it, and those who are leuco-phlegmatic or lymphatic being less susceptible. The condition of asthenia conduces to the production of enteralgia, and a lowered vitalism is often associated with an exaltation of sensibility. The weakness resulting from acute or chronic disease, by depressing the tone of the system in general, and the functional power of the

great nervous centres in particular, constitutes a common predisponent, and the morbid action of the nerves proper to some part or parts is not an unusual occurrence. During the convalescence of fever, after visceral inflammation and large losses of blood these attacks are not infrequently witnessed. Excessive lactation, by subduing the general strength, often enters as an element into the remote causation; and the same may be said of menorrhagia, the lochia, hæmorrhoids, leucorrhœa, and like affections. Long-continued secretions and periodical discharges, by deteriorating and diminishing the vital fluids, are followed by the result in question.

Amongst the proximate or exciting causes is to be mentioned the malarial influence, and in tropical countries and aguish districts there is no doubt it often merits the accusation. Atmospheric humidity, low and damp situations, and a naturally cold and wet climate, form endemic conditions which foster the development of neuralgic ailments; and the truth of the converse is unquestionable that in places of greater altitude and in a purer and dryer air they are not so prevalent. When hot and sunny days are followed by frosty nights, the body being suddenly chilled, and thus the blood being determined to the internal organs, these anomalous pains are often produced. Wet clothes and wet feet give rise to the same affection. Mental fatigue, as after long-continued and great intellectual efforts, has by some writers been enumerated. In those persons whose vocations are such as to demand a continued strain of thought, or whose hopes and fears are excited by speculation, as in commercial enterprises, or those whose faculties are stimulated by some career of ambition, in all of whom the nervous functions are brought into great energy of action, these neurotic ailments prevail, sometimes being located in one organ or part, sometimes in another.

There are also proximate causes, which are strictly speaking pathological—which are referrible to foregoing and obvious forms of morbid change, especially to those changes which take place in the blood, and which constitute a humoral causation to the nervous phenomena. It has been observed by Simon that central neuralgia arises with the utmost frequency in anæmiated and debilitated persons;[1] and we know how apt it is to follow hæmorrhage, and be associated with malnutrition when no primary structural lesion exists. During the latency of the gouty, and in the rheumatic diathesis, when the *materies morbi* of those respective affections has accumulated in the system, before its explosive decomposition has been evinced by local inflammation and excessive secretional evacuation, its presence may be such as to generate that humoral disorder, which first affects the cerebro-spinal and ganglionic centres, and then the nerves proper to visceral organs. In chorea, which is consequent upon some perversion in the development of the blood, caused by the alteration of physical qualities, or the chemical relations of that fluid, or it may be by the absolute generation of some new product, we have ample testimony of the

[1] Lectures on General Pathology, lect. x.

immediate effect produced on the nervous system. And in Bright's disease we are continually presented with examples of the same consequence, caused by the retention of effete and poisonous matters in the circulation. Dr. Todd[1] some time ago pointed out the fact that epilepsy, as associated with this renal affection, is characterised by greater severity in its seizures the longer the interval between the fits, because the irritant materials revulsed into the circulation are then in accumulation and act with greater force. The fact that defective blood-development, or its contamination by lesion of the depurative organs, is productive of nervous disorders, is well shown by the administration of suitable remedies. In anæmia and chorea we every day observe the beneficial effects of ferruginous medicines, and see how pains diminish in degree and frequency, and how the disorderly movements of the voluntary muscles become subdued. In hyperæmia, more especially in that form which has been denominated active hyperæmia, pressure upon the nervous filaments gives pain ; and although such far more frequently obtains with the solid abdominal organs, yet it doubtless is an element entering into the causation of Enteralgia.

In organic disease of the brain and spinal cord pain is generally reflected to some distant part, and such is the common case in lesion of the last-named organ. In caries of the vertebræ, as I have in repetition observed, the reflected visceral pain has been a constantly recurring sign. Some years ago I saw, at the request of a distinguished provincial surgeon, a lady who for many weeks had been under his care, and whose case he regarded as one of persistent Enteralgia caused by some offending ingesta or some impaction in the bowels. I believed, however, that this pain in the bowels had a more remote origin—that it was spinal. The examination after death revealed vertebral caries and softening of the cord. Sometimes the distal pain can be traced to mechanical injuries of the nerve-centres. We know that in children there is the closest connexion between encephalic disease and disorder of the bowels. In primary disease of the solid abdominal viscera, especially in that of the liver and spleen, irritation is not infrequently extended to the intestines ; sometimes neuralgic pain of an intermittent or remittent character eventuates ; while in active congestion of the liver, or in that sudden distension of the spleen which occurs in periodic fever, intestinal pain is no unusual symptom. The intimate sympathy which subsists between these parts can be well understood when we consider their ganglionic connexion.

Amongst the more common causes may be mentioned indigestion and flatulence. When the ingesta have not been properly converted into chyme, but have passed down into the lower bowels, only partly disintegrated, they give rise to irregular spasmodic attacks of pain by acting, as it were, like foreign bodies in the canal. In this way shell-fish, dried salt meats, pork, badly cooked food, unripe fruit, crude vegetables, and the like, are followed by the affection. That flatus

[1] Lumleian Lectures, *Medical Gazette*, 1849 and 1850.

very often produces Enteralgia is a fact so familiar as scarcely to merit comment; but numbers of the older authors speak of this cause with much emphasis.[1] Wiessner says: "Flatus similiter etiam ventriculum doloribus spasticis afficiunt. Hæc enim toti tractui intestinorum molestissima affectio vel ipsi ventriculo proxime nocet, vel partium distentione stomacho proximarum. Ex hisce imprimis colon transversum, ante inferiorem ventriculi curvaturam extensum, sedem aëri incluso quam maxime incommodam parat."[2] The movement of gases from one part of the intestines to another accounts for the shifting of the pain. Constipation is another and frequent cause of the complaint. Indurated masses of fæces become impacted in the cæcum, sigmoid flexure, or transverse colon, and attacks of sharp, twisting, rolling pain come on from time to time, and are not permanently relieved until the irritative contents of the gut have been voided. Sometimes a large gall-stone or a concretion is the cause. Morbid secretions, acrid substances, acerb fruits, septic food, such as putrid game and bad cheese, stimulating liquors, and sour drinks are liable to produce Enteralgia. Chemical agents and medicinal compounds are followed by a like result. The sensitive fibriles proper to the lining tunic of the digestive tract, by coming in contact with the fore-named, become irritated, and there may be great pain when the motor nerves are but slightly influenced in their functions. In lead-poisoning the intestinal nerves are particularly prone to exaltation of sensibility.

SYMPTOMS.—The mode of accession is generally sudden, the pain being sharp, shooting, or twisting; but in some instances it comes on more gradually, and a rolling or aching of the bowels is described. The affection is in the majority of cases first felt at the umbilicus or in the right iliac fossa. The paroxysms increase in degree and frequency, the intervals from suffering being irregular and of varied duration. The pain, especially in the earlier stage of the attack, alters its position. It is rather relieved than aggravated by pressure. The skin is often cool, the face pale, and the pulse, instead of being accelerated, is rendered slower than natural. In the severer cases the stomach sympathises, and sickness and vomiting may supervene; and when the malady becomes intensified and the agony excessive, the entire surface is bedewed with a chill, clammy perspiration, the extremities becoming cold and of venous hue, and the general aspect that of collapse. Costiveness is the common accompaniment, and percussion displays an overloaded state of some part of the colon, generally at the cæcum or sigmoid flexure. When flatus is the chief cause, there is intestinal distension, and such notably obtains in the large bowel. On palpation nodulated eminences are felt, which

[1] Rhodii Obs. Med. cent. iii. Palav. 1657; cent. ii. obs. 70. Lieutaud, Hist. Anat. Méd. tome i. p. 7; Paris, 1767. Marchand, Diss. de Cardial. flatul. Argent. 1754. Weikard, Vermischte med. Schriften, Frankf. 1778, b. ii. p. 143.
[2] De Spasmo Ventriculi, p. 13.

quickly alter in their configuration, and which are caused by the constricted and distended portions of the tube. With the expulsion of the confined gases the patient derives signal and immediate relief, and sometimes the amount evolved is very considerable. The noisy flatulent movements—borborygmi—which are often heard in the canal frequently constitute a marked symptom in hysterical females whose primæ viæ are generally disordered, and whose assimilative functions are imperfectly performed. The attacks of Enteralgia may be intermittent or remittent. Sometimes they terminate with all the rapidity with which they were ushered in. Although, as a rule, Enteralgia is apyrexial in character, yet inflammation sometimes occurs; and then the surface is warmer, the pain more fixed, and the circulation excited. In hysterical women uterine disorder is the usual concomitant, and the enteralgic pain will often be found in association with spinal tenderness. In such instances percussion on the spinal processes should not be omitted, and not infrequently hyperæsthesia of the abdominal surface is a prominent sign. When the subjective symptoms are referrible to organic disease, and are evidently reflected, the cerebro-spinal axis and the solid abdominal organs should respectively be examined, and the kind of lesion there existent be as far as possible correctly estimated. This neuralgic pain of the intestines is occasionally seen as a symptom caused by ulceration and congestion of the uterus; and it may come on after the sudden retrocession of cutaneous eruptions and the exanthemata; also, as before remarked, it may follow profuse critical evacuations, the repeated loss of blood by hæmorrhoids, or other sources of debility.

The symptoms are modified or terminated, or the attacks rendered less recurrent, by the accession of certain morbid conditions taking place in the system. The advent of a powerful diaphoresis, the supervention of diarrhœa, the flow of the catamenia, the lochial discharge, the occurrence of epistaxis, the formation of an abscess, or the return of some long-habituated secretion, are known to exert such influence. A fit of gout, or the development of acute rheumatism, seem on derivative principles to lessen this nerve-pain, and diminish in or remove from the organism those conditions of irritation which particularly affect the cerebro-spinal and ganglionic nerves. It sometimes happens, when the affection comes on in females, that a very large secretion of pale or almost colourless urine is at once succeeded by the mitigation of the attack. In some cases the abdominal and thoracic muscles are spasmodically contracted; there are rigidity of the recti and a loss of motor power in the intercostals, the chest is fixed, and the breathing is oppressed. When the seizures become repeated, they are liable to be characterised by greater severity, the exaltation of the nervous sensibility doubtless becoming augmented by continued irritation. The duration of the symptoms is always most uncertain, as much will depend upon the kind of fundamental cause by which they are produced: it may, however, be regarded as the most usual fact that the more severe the fit the shorter will be its continuance.

In children spasmodic pain of the bowels is soon productive of disorder in the digestive functions, and irritation in the alimentary tube is soon followed by the ordinary conditions which characterise infantile convulsions. According to M. Billard, the child cries suddenly and loudly, the face is contracted, the limbs are stiffened, the belly is tender to the touch, there is tympanitic distension, and the attack is often relieved by the expulsion of large quantities of gas *per anum*.[1] The alvine evacuations are generally suspended, and frequently there are vomiting and carpo-pedal contractions; in young infants there is tossing of the arms, the legs are drawn up to the abdomen, and often in the course of time green and offensive stools are voided. Frequently upon investigation it will be found that the mother's milk, or the artificial food which has been given, is the cause.

PATHOLOGY.—In the discussion of this part of the subject, those morbid conditions may first be noticed that consist of impairment of the functions of the bowels, which are characterised by alteration of sensibility, and which are often in association with a lowered state of vitality in the economy; but, as in all functional affections, the real origin of the complaint cannot always be detected, and remains an uncertain inference. It frequently happens that when some irritation of the mucous surface of the bowels is the cause, gases become generated, and painful dilatation of some part or parts of the tube is the consequence. It is probable that a great portion of the gas is secreted from the blood, for the flatus is often produced too quickly and in too great abundance for the presumption that it comes entirely from the decomposition of the ingesta. By this distension of any particular section of the gut there is loss of tone, the contractile power of the muscular coat may be almost or entirely abolished, and the pressure on the sensitive nervous fibriles occasioned by such dilatation will well account for the complaint, because there may be asthenia of this part of the ganglial system in accompaniment with morbid exaltation of sensibility. This irritation of the peripheral nerves, caused by harmful ingesta, concretions, vitiated secretions, and the like, affects not only the intestines themselves, but other organs also; and thus it is that the heart and diaphragm are functionally influenced, and hence the depressed circulation and difficult respiratory movement so commonly witnessed in the more violent examples of the ailment. As pain is often to be regarded as the prominent expression of some malady pervading the entire system, and as the functions of any organ may be thus disturbed, it not infrequently occurs that one or other of the viscera is the seat of such disease; and thus it is that in contamination of the blood spasmodic or neuralgic pain of the intestines may result. In saturnine poisoning there is ample illustration of this fact; the poison is transferred into the circulation, the secretions are arrested, and fits

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[1] Traité des Maladies des Enfans nouveaux-nés et à la Mamelle. 8vo. Paris, 1828.

of agonizing pain are felt in the bowels : and so it doubtless occurs in those dyscrasial affections in which the fluids of the body are degraded by changes more occult, and by the operation of agents less plainly comprehended. In gout and rheumatism, and in Bright's disease, the cerebro-spinal and organic centres are secondarily influenced through debasement of the blood. When gout is retrocedent or rheumatism suppressed, their peccant materials are revulsed upon and irritate some of the internal organs; and in Bright's disease, when the urinary excreta are imperfectly eliminated, the disturbance of the nervous system is exemplified not only in the exaltations of sensibility in the viscera—neuralgia—but in perverted motor function, as evidenced in the reflex action of vomiting and diarrhœa. In simple asthenia, when there is excess of emotional and other motility, as in hysterical females, the kinds of pain in question are readily developed; and if it cannot be said that structural changes do not exist, at least they cannot be indicated.

When organic disease in some cognisable form does constitute the cause of Enteralgia, the examples may be most varied in their locality, degree of objective symptoms, and the kind and amount of their structural alteration. When the primary lesion is in the cerebro-spinal axis, chemical, physical, and mechanical aids to diagnosis are of no avail, and we are compelled to rely upon analogies and subjective representations.[1] One of the most frequent pathologic conditions is that of hyperæmia, which produces irritation and reflex phenomena. According to Brown-Séquard,[2] when the afflux of blood or other morbid change is at the posterior parts of the cerebro-spinal axis, hyperæsthesia is the common result; and it would seem that interruption of continuity of the vaso-motor nerves is the fundamental cause of vascular dilatations. In spinal irritation and hysteria reflex visceral pains thus doubtless arise; and it may truly be affirmed that the causes of spasmodic and neuralgic pains are more commonly central than peripheral—in figurative language, they are more frequently referrible to the battery than the conducting wires. In positive inflammation of the cerebral and spinal tissues abnormities of function must necessarily arise. The nerves are seldom diseased. Albers and West in only exceptional cases found the vagi morbid on inspections after hooping-cough. Bichat[3] repeatedly examined the nerves in diseases of the viscera without discovering pathologic change. But according to the testimony of various writers, and from my own observations, the nerves are sometimes inflamed, and are subject to other morbid alterations. The neurilemma is the part most prone to inflammation, and Craigie[4] asserts that such condition is a common cause of neuralgic pain. In tetanus and sciatica the entire nerve has been seen red and swollen. The sympathetic ganglia are sometimes diseased; there may be vascularity of the cellular tissue

[1] Sieveking, Manual of Patholog. Anat. p. 211.
[2] Lectures on the Central Nervous System, p .205.
[3] Anatomie générale, i. 225.   [4] Pathological Anatomy, 2d edit. p. 380.

interposed between the elements of the ganglia, and the ganglionic substance has been seen enlarged and indurated. It is also highly presumptive that there may be molecular change in the contents of the ganglionic corpuscles. Neuromatous formations may be the cause of Enteralgia. As I have already remarked, the more common and obvious conditions of visceral disease may produce enteralgic pain, such as thickening of the inner tunics of the bowel, whereby impediment is given to the contents of the tube; an ancient band of lymph giving rise to constriction; or by some abnormal growth pressing upon the gut. And in the various organic affections to which the solid organs are liable, reflected enteralgic pain is no unusual result. In diseases of the urinary and generative organs of both sexes the kinds of abdominal pain now spoken of not unusually supervene. Ulceration of the uterus and impaction of the ureters sometimes cause Enteralgia.

DIAGNOSIS.—The diagnostic indications of Enteralgia are sudden, darting, plunging, or twisting pains, which come on paroxysmally, the attacks varying in their degree of severity and in their duration. The intervals between the seizures may be almost or altogether free from suffering. The pulse remains unaltered, the surface is cool, and the facial expression is that of pallor and pain. There is moist tongue, no thirst, the bowels are confined, and flatulent distension is the common accompaniment. Pressure on the abdomen relieves rather than augments the pain, and it is not unusual for the patient to press his hands on his belly during the paroxysm as a means of affording relief. The expulsion of gases from the large bowels gives immediate ease; and sometimes the advent of diarrhœa at once cuts short the complaint. In inflammation of the bowels pressure confers pain, the skin is hot and dry, the pulse quick, the face flushed, the secretions and excretions are diminished, the patient cannot turn and twist about in bed as he can in Enteralgia, and the objective symptoms of symptomatic fever are more or less proclaimed. In inflammation the pain is confined to one particular part of the abdomen, and only gradually becomes diffused. In Enteralgia it shifts about with great celerity. In ileus there is vomiting, and at length of fæcal matters; a lump can often be felt, and the suffering, as in inflammation, does not intermit. When this neuralgia of the bowels is from impaction of fæces, palpation and percussion will be our guides; if from concretions or mechanical obstructions, the history of the case and collateral circumstances will conduct to a right decision; and if from irritative secretions, a flux generally supervenes. If reflected by distal disease, as in hepatic, splenic, and renal ailments, those organs should be carefully examined. In neuralgia the pain radiates round to the back, generally at one side. In the passage of renal calculus the pain is in one side; it darts down towards the pubes and thigh, and in the male there is retraction of the testicle. In rheumatism of the abdominal muscles the disease pervades some other part. In hysteria the spine

should be examined; and when from this cause, often a copious discharge of colourless urine will give relief. In lead-poisoning there will mostly be dropping of the wrists, and the blue line on the gums.

TREATMENT.—The remedies first indicated are those which are most likely to abridge and mitigate the sufferings of the paroxysm; and with this view antispasmodics and anodynes may be prescribed, such as opium, chloric æther, henbane, conium, camphor, ammonia, and similar agents. At the same time hot fomentations, sinapisms, terebinthinate epithems, or stimulating and rubefacient liniments, may be employed. The surface should be kept warm and diaphoresis promoted, which can be best accomplished by the patient first putting his feet and legs into hot mustard and water, and then going to bed. The warm bath and sedative enemata are excellent auxiliaries. Sometimes anodyne embrocations, addressed to the spine, do much good. In the more chronic neuralgic affections, I have long been in the habit of prescribing a liniment composed of laudanum, chloroform, the extract of belladonna, and the linimentum camphoræ. The bowels should afterwards be cleared out by mild laxatives, such as castor-oil, the compound rhubarb pill, extract of colocynth in combination with extract of henbane, or the galbanum pill, or the confection of senna. When we believe the fundamental cause to reside in the solid viscera, *or* in the cerebro-spinal axis or ganglionic centres, our measures should then be addressed to such parts, and our aim be to lessen the general morbid excitability of the nervous system.

ENTERITIS.

By John Syer Bristowe, M.D. F.R.C.P.

THE term Enteritis, signifying inflammation of the bowels, is of ancient date, and from the earliest times until now of more or less loose and various application. It has often been applied to a certain group of symptoms irrespective of the conditions under which they may arise, and irrespective even of the presence or absence of actual inflammation, as for instance to strangulated hernia, intestinal stricture, and other forms of obstruction of the bowels; and again the word has often been made to include various specific forms of disease attended with specific intestinal lesions, such for example as enteric fever, tuberculosis, and cancerous infiltration. It is intended in the present article to treat of Enteritis, according to its real meaning, as a simple inflammatory affection; and to eliminate from the subject, as far as possible, all reference to the diseases with which it may be confounded or on which it may supervene.

I. As AFFECTING THE SEROUS AND MUSCULAR COATS.—The intestinal tunics are all of them liable to inflammation either separately or in combination: and the inflammatory process, as it occurs in each, has a tendency to present characteristic peculiarities, and to be associated with special symptoms. Inflammation of the serous coat is of frequent occurrence as a part of general peritonitis, a disease the morbid anatomy and symptoms of which are subsequently described; but, as in the analogous cases afforded by the pleuræ and pericardium, inflammation commencing here spreads rarely, or with difficulty and late, to the subjacent tissues; and hence peritonitis may be considered practically to be as distinct from true inflammation of the bowels, as pleurisy is from pneumonia, or pericarditis from inflammation of the heart. Nevertheless inflammation beginning at the peritoneal surface does occasionally invade the whole thickness of the intestinal walls; and still more frequently, just as pneumonia induces inflammation in the overlying tract of pleura, inflammation of the deeper tissues of the bowel leads to circumscribed inflammation of the investing peritoneum, and to the superaddition of peritonitic symptoms to symptoms previously existing. The structures lying between the serous and mucous tunics, namely, the muscular laminæ with their associated nervous plexuses and connective tissue, are

rarely the primary seat of inflammation; occasionally, it is true, in pyæmia, and under other exceptional conditions, an abscess forms in them; but they are more frequently involved in the extension of peritoneal inflammation; and still more frequently they become inflamed either by the spread of inflammation from an inflamed mucous membrane, or in consequence of its simultaneous origin in the several intestinal tunics. Inflammation and its results here, in their slighter forms, scarcely reveal themselves to ordinary post-mortem examination, but when more pronounced are manifested anatomically by congestion and effusion of serum, lymph, pus, or blood. The symptoms which they induce are in the first instance probably spasmodic contraction of the muscular fibres, subsequently loss of power or complete paralysis.

II. As affecting the Mucous Membrane.—Inflammation as a primary and characteristic affection occurs far more frequently in the mucous membrane than in the coats external to it; and it occurs here in forms which vary considerably according to its cause, the constitutional conditions under which it arises or with which it is associated, and its degree of intensity.

(*a*) *Catarrhal Inflammation.*—The slightest and simplest form of inflammation is usually termed *catarrhal*. This may be produced by the local action of irritating ingesta, or by the influence of those external conditions which are known to be the agents in setting up the same kind of inflammation in other parts; and it is believed by some to attend generally scarlatina and other specific fevers.[1] Young children, particularly during the period of teething, seem specially liable to it. It is characterised by congestion, tumefaction, softening and dryness of the mucous membrane, followed speedily by the secretion, often in considerable abundance, of mucus, which is ropy or watery, irritating, and sometimes mixed with blood. It sometimes affects the lower bowel only, producing mild dysenteric symptoms; but frequently it commences in the upper bowel, or in the stomach, and spreading thence downwards gradually traverses the whole of the intestinal canal, causing in its progress more or less uneasiness, aching and griping, attended frequently with nausea and sickness while it is still high up, with diarrhœa and expulsive pains and efforts when it reaches the large intestine. The tongue is generally more or less furred and dry, the breath offensive, and the appetite impaired; but these symptoms vary, and are often absent, especially when the large intestine alone is affected. Some degree of general febrile disturbance, indicated by heat and dryness of skin with sense of chilliness, increased frequency of pulse, lassitude and headache, is usually attendant on the local disorder. In children, in whom inflammatory affection of the gastro-intestinal mucous membrane is sometimes associated with aphtha, the disease not infre-

[1] See Dr. Fenwick on "The Condition of Stomach and Intestines in Scarlet Fever," Med. Chir. Trans. vol. xlvii.

quently produces serious results and death, either from the debility
which follows persistent diarrhœa and vomiting, or from the superven-
tion of cerebral complications, such as convulsions or coma. There can
be no doubt that a large number of cases of gastro-intestinal dis-
turbance and of diarrhœa are due to catarrhal inflammation ; yet the
existence of such inflammation is more a matter of inference from
symptoms than of direct observation upon the condition of the mucous
membrane. For the latter can only be examined after death, at
which time congestion and other indications of superficial and slight
inflammation have for the most part disappeared, or are lost in
post-mortem changes.

(b) *Croupous Inflammation.*—The designation "croupous" (diph-
theritic or membranous) inflammation is given to those cases in which
the mucous surface becomes covered to a greater or less extent with
a more or less adherent membranous film, consisting of corpuscular
elements cemented together by a coagulable exudation, and prolonged
for the most part by rootlets from its under-surface into the Lieber-
kuhnian follicles. This affection, which is far from uncommon,
may sometimes doubtless be regarded as the expression of some
specific form of inflammation ; certainly many believe (and I am one
of them) that it is a common feature in the early stage of dysentery ;
at the same time it frequently occurs quite independently of all in-
fectious or malarious influence. It undoubtedly indicates greater
intensity of inflammation than mere catarrhal inflammation ; there is
generally much greater congestion and thickening of mucous mem-
brane, and not infrequently hæmorrhage, suppuration, or gangrene.
Croupous inflammation is often met with in the large intestine in
scattered patches, which are sometimes linear, sometimes irregularly
polygonal or stellate, and occupy for the most part the prominent
ridges of the mucous membrane, more especially the edges of the
intersaccular constrictions. In some cases, still chiefly occupying the
more prominent parts, it forms a coarse, irregular network extending
over large tracts of surface ; in other cases it forms uniform patches
of considerable extent. It is less common in the small intestines, but
may be found in them affecting the free edges of the valvulæ con-
niventes, or spread over a large area. It is sometimes met with on
the surface of tracts of cancerous infiltration which are on the eve of
ulcerating. It may be added here that cases sometimes come under
observation in which patients pass *per anum* shreds of false mem-
brane, or even membranous casts of the bowel, of soft texture, various
thickness, and of a dirty greenish or brownish hue. This discharge
is generally, if not always, a consequence of dysenteric ulceration.
The symptoms which attend croupous inflammation are not special ;
they vary, according to circumstances, on the one hand between those
of diarrhœa and dysentery, and on the other hand between those of
mere colic and of typical enteritis. The patchy form, indeed, so
common in the large intestine, is often overlooked during life, from
the fact that it occurs as a complication in the later stages of many

e disorders, as for example acute pneumonia, Bright's disease,
nosis of the liver, and cerebral affections.

) *Chronic Inflammation and Degeneration.*—Both catarrhal and
pous inflammations, in their slighter degrees, generally, and for
most part speedily, undergo resolution. Sometimes, however,
end in ulceration; an event which, with its consequences, is
considered further on. And sometimes they lead to persistent
ifications of the mucous membrane which are often included in
term "chronic inflammation." These consist generally in slight
lensation and hardening of the mucous tissue, more or less dis-
t congestion, or black pigmentary deposit in the villi and inter-
cular spaces, some degree of atrophy of the Lieberkuhnian
cles, and granular or fatty degeneration of their epithelial contents,
ther with an analogous condition, more or less pronounced, of
epithelium of the mucous surface generally. The solitary and
inated glands are sometimes atrophied, sometimes larger and
e obvious than natural. The changes indeed are chiefly changes
egeneration; and in that sense, as probably also clinically, are
ted to the lardaceous degeneration which occasionally happens
ersons labouring under chronic tuberculosis, bone disease attended
suppuration, and secondary syphilis. Lardaceous degeneration
rs later in the bowel than in the liver, spleen, and kidneys; it is
d chiefly in the lower part of the ileum and in the large intes-
; it affects in the first instance the small arteries and capillaries
nd, and in, the solitary and agminated glands, which bodies
me swollen; and then gradually tends to involve the whole thick-
of the intestinal wall, the muscular fibres and other tissues
ming finally infiltrated. The bowel thus becomes thickened, and at
same time harder than natural; and often in the later stages erosion
he affected glands occurs, leading in Peyer's patches to a reticu-
d condition of surface. The above chronic affections of the
cous membrane are generally associated with diseased conditions of
r organs, to which indeed they are secondary; and not infrequently
stomach is at the same time the seat of some chronic morbid
ess. The presence of these complications, and the fact that
ically ulceration of the bowels, together with tubercular and other
bid processes, passes in a large number of cases for chronic inflam-
ion, render it difficult to isolate the clinical phenomena due
ially to the bowel affections now under consideration. They
btless vary greatly; but may be briefly summarized as com-
ng in various proportions, both relatively and positively, imper-
digestion of the alimentary matters received into the intestine,
ssive secretion of more or less watery mucus, increased peris-
ic movements with griping pains, looseness of bowels with dis-
rge of watery, or yeasty, or otherwise unhealthy and offensive
uations, and innutrition from the imperfect absorption of food.

ee a good account of lardaceous degeneration. M. Hayem, quoted in New Syden-
Society's Biennial Retrospect of Medicine and Surgery, for 1865-6. p. 176.

III. AS AFFECTING THE WHOLE THICKNESS OF THE BOWEL.—
By the older writers generally, and for the most part also by those
of more recent times, the simple unqualified name "Enteritis" has
been used to signify a special group of symptoms associated with
the presence of a more or less extensive tract of intensely inflamed
bowel. The affection here referred to is termed by Cullen phleg-
monous enteritis, in contradistinction to the milder varieties of
inflammation, affecting the mucous membrane only, which he in-
cluded under the name of erythematous enteritis.

The symptoms, which are supposed to characterise this form of
enteritis, may creep on insidiously or show themselves in sudden
intensity, and consist mainly, in the earlier stages, in more or less
severe abdominal pain (resembling in its character and in its increase
by pressure and by movement the pain of peritonitis, but differing
from it in being associated with colic), obstinate constipation, nausea
and vomiting (occurring both after and independently of the ingestion
of food), and marked febrile disturbance; and subsequently (supposing
the case to be going on unfavourably) in the gradual supervention
of tympanitis, attended, for the most part, with diminution or even
total cessation of abdominal pain and tenderness, with still persistent
constipation and vomiting (the vomited matters becoming opaque,
brown, and fœtid, if not actually fæcal), with hiccough frequently,
and with collapse (indicated by extreme feebleness of pulse, cold-
ness and dampness of the surface, especially in the extremities), and
finally death from asthenia. The morbid changes which may be
looked for after death are such as are produced by intense inflam-
mation of a limited tract of intestine. The affected part, which is
mostly in the small intestine, and which may vary in length from an
inch or two to one or two feet or more, is as a rule much dilated; its
serous surface presents a general dusky red, or slate, or purplish-black
colour, due to the condition of the parts internal to it; it is marked,
too, by lines or patches of more or less intense superficial congestion,
may present blotches of sub-serous extravasation, and is often covered
more or less with adherent lymph; its mucous and sub-mucous tissues
are mostly somewhat thickened and softened, sometimes only mode-
rately congested but presenting spots and streaks of extravasation,
sometimes black from combined congestion and extravasation, some-
times pale and infiltrated with lymph or pus, sometimes distinctly
gangrenous; and its middle coat, sharing in these changes, is also
more or less swollen and soft, and congested or œdematous, or the
seat of some form of inflammatory exudation. The inflamed tract
usually presents fairly well-defined limits, terminating abruptly below
in pale and healthy but contracted and nearly empty bowel, above in
bowel which may also be healthy, but is dilated like the diseased
portion and filled like it with fæcal contents. The diseased intestine
contains frequently in addition to simply fæcal matters more or less
sanguineous exudation; and traces of the same exudation may often
be discovered in the contracted bowel below.

Now, the above phenomena are by no means infrequently met with; they are the common accompaniments of strangulated hernia and of intussusception; they are present in those cases in which, as is supposed, the sigmoid flexure or some other loop of bowel becomes twisted on itself and thus strangulated; they supervene whenever a gall-stone or other foreign body of sufficient size becomes fixed in its passage along the intestine; they occur sometimes also as a late event in stricture, or in those cases in which the bowel becomes constricted by bands of lymph; they are sometimes developed as a result of the extension of inflammation, either from peritoneum or from an intestinal ulcer; and very rarely indeed they originate idiopathically, that is to say from such general causes as produce idiopathic peritonitis, idiopathic pneumonia, and the like. Enteritis, therefore, is a disease which is almost always complicated with some other grave lesion, on which indeed it depends, and which modifies alike its symptoms and its progress.

But even in the uncomplicated form of the disease, which is alone now under consideration, the symptoms are liable to considerable variety; the variations depending mainly on the degree of intensity of the inflammation and its extent, and on the situation of the affected portion of bowel. Indeed, the two principal factors in producing the characteristic symptoms of enteritis are inflammation, on which depend the various febrile phenomena, and paralysis of the inflamed portion of bowel, which permits of its passive dilatation by the accumulation of contents, opposes a more or less complete bar to their transit, and thus induces on the one hand constipation, on the other vomiting.

The most important practical distinction between colic and enteritis, is, according to most authors, the absence of febrile symptoms in the former disease, their presence in the latter. And no doubt in most cases of enteritis febrile symptoms manifest themselves in a marked degree, at least in the earlier stages of the malady. Heat of skin, rigors, quickness and hardness of pulse, not infrequently mark the onset of the attack; but it is a mistake to suppose they are always present, or at all events readily perceptible, for in many cases no rigors are experienced, and in some there is little or no acceleration of pulse until towards the close of life, and no more heat of surface than may attend, and often does attend, the gripings of ordinary colic. There is mostly some dryness and clamminess of mouth, if not absolute thirst; and the tongue, which is occasionally pretty clean at the beginning, becomes generally soon thickly coated and ultimately dry. Another feature of enteritis upon which much reliance is placed is the association of the abdominal pain and tenderness of peritonitis, with the tormina of colic. Pain and tenderness are certainly present in most cases, at least in the beginning, and in dependence upon them the dorsal decubitus, so characteristic of peritoneal inflammation. But these symptoms vary greatly; sometimes they are intensely severe, sometimes they are from first to last scarcely

appreciable, and generally they subside in the progress of the case. It can readily be understood that when the peritoneal surface is largely involved, the pain and tenderness will generally be proportionably severe; that when an extensive length of bowel is affected, there will be correspondingly extensive uneasiness and tenderness; and that when, as sometimes happens, the serous surface is not inflamed, or when the affected portion of bowel is small, the pain and tenderness may be not only limited in extent, but no greater than one finds them in colic or in simple ulceration of the mucous membrane. It is worth while to remark, that limited pain and tenderness are very commonly referred to the region of the umbilicus. Tormina are often at the onset very agonising, being then probably due in some measure to the spasmodic movements of the inflamed bowel; but they continue even after paralysis has become established, in consequence of the violent but ineffective efforts of the bowel above the seat of disease to overcome the impediment which the disease produces. But tormina are sometimes scarcely recognisable, and frequently, like pain, cease comparatively early. Constipation and vomiting are among the most essential symptoms of enteritis. In the uncomplicated affection the impediment to the action of the bowel is due simply to the presence of a paralysed and inactive zone of greater or less breadth between an upper and a lower length of healthy bowel; it is no necessary part of the disease, therefore, that the outbreak of acute symptoms shall have been preceded by constipation, or even that after the disease has become established the portion of the bowel below the inflamed part shall not empty itself; and, it may be added, that in a variable degree the contents even of the inflamed gut may slip or be squeezed onwards into the healthy tube beyond, and that even calomel, and such other purgatives as act rather through the system than directly, may produce to some extent their characteristic effects. But it is nevertheless a fact that the inflamed bowel is really a substantial impediment, that there is therefore during the progress of the disease marked constipation, and that purgatives as a rule produce no purgative effect. Vomiting may occur in colic, in diarrhœa, in simple peritonitis, and in many other conditions as a mere sympathetic affection; and sympathy has probably some share in its production even in enteritis, at least at the commencement. But ultimately the vomiting here is due directly, like the constipation, to intestinal obstruction. In the first instance, no matter where the obstruction or what the immediate cause of vomiting, the vomited matters are merely the secretions of the stomach mixed with alimentary substances; but soon bile becomes mixed with these; and before long glairy mucus and bile alone are discharged. Then the eructations become fœtid; and soon the fluid brought up gets turbid and brownish, and by degrees comes to resemble the contents of the lower part of the small intestine; but it becomes fœtid also, and sometimes much more fœtid than the contents of a healthy bowel ever are, the fœtor being caused partly by decomposition of the fæcal matters, partly, as in dysentery, by the

discharges taking place from a gangrenous or otherwise diseased mucous surface. This vomiting of the contents of the intestines is, as Dr. Brinton has well explained, not due to inversion of peristaltic action; but is the result of the gradual accumulation of matters in the bowel above the seat of disease, of their mixture gradually effected by the normally-directed peristaltic movements of the bowel, and of their escape into the stomach partly by simple overflow, induced sometimes by mere change of posture, partly by the pressure exerted on the distended bowel by the surrounding viscera, and by the muscular walls of the abdomen. The fœtid matters which thus reach the stomach often, towards the close of life particularly, escape from it into the mouth by mere regurgitation. Tympanitis is probably in no case wholly wanting; in an early stage it may be, and perhaps usually is, absent or but little marked; ere long, however, the abdomen begins to enlarge, and generally as the case progresses becomes greatly distended, tense, and drum-like. This condition is of course mainly due to the distension by fæcal contents and flatus of the portion of intestinal tube which is inflamed and of that which is above it, but now and then it is connected with rupture of the distended intestine and escape of gas into the peritoneal cavity—an accident, it need scarcely be said, of fatal augury. The pulse at the beginning is, as has been already remarked, often accelerated and hard, but it varies greatly in different cases, both in frequency, volume, and strength, and sometimes retains pretty nearly its ordinary healthy character throughout at least the earlier stages of the disease. As the fatal issue, however, approaches, it becomes more and more feeble, and sometimes at length wholly imperceptible at the wrist; it generally becomes then also quicker, sometimes slower, and not infrequently irregular. The temperature of the skin is usually in the first instance more or less elevated, and its surface dry; but even then perspirations are apt to break out, especially during the paroxysms of colicky pain: subsequently, however, the temperature falls, the extremities and face become cold and pale, or livid, with sometimes a faint tinge of jaundice, and all parts of the surface bathed in profuse cold perspiration. The expression of the patient is generally indicative of anxiety and distress, and it has often been noted that, towards the close of life, the face becomes pinched and shrivelled, and assumes an unnatural aspect of old age. He generally retains his senses throughout his illness, and even up to the moment of death; but this event is often preceded by a period of quiescence or lethargy, and occasionally by slight rambling and almost complete unconsciousness. It may be added here, that there is generally in enteritis more or less complete suppression of urine, a phenomenon which has been variously interpreted, but which is probably due, as Mr. Sedgwick[1] argues, to the influence of the abdominal sympathetic system.

Enteritis, in that intense form of it which has been now described, is undoubtedly a very fatal, and indeed very rapidly fatal, malady.

[1] Med.-Chir. Trans. vol. li.

It is so difficult, however, practically to isolate the comparatively few cases in which it forms the primary and sole disease from the many in which it supervenes as a complication of some pre-existing graver lesion, that the former scarcely admit of statistical examination. As respects the duration, however, of fatal cases, it may be asserted that it rarely exceeds a week, and that it may be as short as twenty-four or thirty-six hours.

IV. TREATMENT.—It seems scarcely necessary to discuss here the treatment of simple catarrhal and croupous and chronic inflammation of the bowels; these inflammations, indeed, are so intimately connected, on the one hand with inflammatory conditions of the stomach, on the other with dysentery and diarrhœa, which have all been elsewhere described at length, that the reader may be safely referred to the articles relating to those diseases for the principles and details of treatment applicable to the inflammations now in question.

In reference to the treatment of the more severe forms of enteritis, two main principles seem now to be fairly well-established : they are, first, to relieve pain, and prevent, so far as may be, all movements of the bowels, by means of opium; secondly, to avoid every attempt (at least until all grave symptoms have ceased) to force the bowels by the administration of purgatives. It has been shown quite conclusively, principally by the experience derived from the after-treatment of strangulated hernia, that it is always dangerous to endeavour to propel fæcal matters through an enteritic length of bowel, that in most cases the effort is useless so far as their effectual propulsion is concerned, while, by the augmented muscular and excretory action which is thus produced in the bowel above, the diseased tract below becomes more and more distended, almost certainly more and more softened, congested, and inflamed, not infrequently becomes ruptured, and at the very least has its progress towards recovery delayed. Besides which, purgatives tend greatly to increase pain, and vomiting, and general distress. And, indeed, when one considers the great length of time during which constipation may continue with little or no influence on the general health, how long patients with impassable stricture of the bowel manage often to survive, it must be obvious that the constipation of a disease of so short duration as enteritis is not of itself a grave source of danger. Clearly, if the patient is to get well, his recovery must in the first instance be dependent on the recovery by the diseased bowel of its healthy tone, and capability of peristaltic action : and to this end our efforts must be directed. But experience shows us that we have little or no power to arrest internal inflammation, unless it be indirectly by promoting the quiescence of parts, and by relieving pain and irritation ; and, for these purposes, opium, in large and frequent doses, is generally our most valuable agent. No absolute rule can be laid down with regard to the quantity of opium which should be given for a dose, or to the frequency with which the dose should be repeated; the patient should, however, be got well

the influence of the drug, and should be kept under its
ice. But the constant vomiting and the distension of the
above the seat of disease, form a serious, if not fatal impedi-
to the absorption of opium received into the stomach; what
llowed may be wholly vomited, or, if retained, very partially
at all received into the system. If therefore it be thought
o administer opium by the mouth, it should be given in the form
iable to provoke, or to be rejected by, vomiting; but it is cer-
best to administer it in the form of suppository or enema, or
ict it subcutaneously. But, no doubt, it is generally desirable,
ven necessary, to associate with the use of opium other details
itment. The question of the abstraction of blood, formerly so
r employed in the treatment of internal inflammations, is not
ly to arise; and it must be acknowledged that there are
in the early stage of which removal of blood may be advan-
s. When, at the commencement of enteritis, the symptoms
toneal inflammation are strongly pronounced, there is no doubt
ie application of twenty, thirty; or more leeches to the surface of
domen is generally followed by great and immediate relief, if not
ual benefit. Doubtless, the removal of blood from the arm would
least equally beneficial; and in cases in which, at the same stage,
nitic symptoms are less distinct, but in which there is high
I should not hesitate to have phlebotomy performed. Warm
ght applications to the surface of the belly generally soothe,
if they produce no further beneficial effect; and sometimes
rd-plasters, and similar mild counter-irritants, give relief.
e same way, enemata of warm water or of warm gruel are
ies useful. There are few symptoms more distressing to the
t than the persistent nausea and vomiting from which he
i, and few therefore which we feel more anxious to relieve; but
are none which, at all events at certain stages of the disease, are
ider the influence of direct treatment. At an early period, when
symptoms are merely sympathetic, ice, hydrocyanic acid, alka-
ime-water, bismuth, carminatives, and other remedial agents,
io doubt restrain them to some extent; and again, when the
e has begun to take a favourable course, they subside naturally,
it any special treatment; but when the vomiting is simply the
of over-distension of the stomach and bowels, to which over-
sion there is no other channel of relief, medicine ceases to have
wer over it. The extreme prostration which so early manifests
is a strong indication of the need of food and stimulants; but
in they be administered with even a chance of benefit? Their
tion by the mouth tends to promote sickness, tends also to
the distension of the already too much distended stomach and
i, while probably, from various causes, little or nothing of them
es absorbed. It is obvious, indeed, as is insisted on by Dr.
n,[1] that alimentary matters, if given by the mouth, should only

[1] Intestinal Obstruction. 1867.

be given in very small quantities, and in a form suitable for their ready appropriation by the system. They may, however, be given in much larger quantities, and with none of the above ill effects, and also with a much greater chance of benefit, in the form of enemata. It is not intended by the above remarks to discourage all attempts to restrain sickness, or to supply stimulants or food; for there are cases which seem hopeless, in which, nevertheless, the bowel is recovering, and in which the alternative of life or death depends upon the judicious use of remedies and of regimen; but only to discourage persistence in lines of treatment when their effect on the patient, and the progress of the case, prove their inutility or harmfulness.

OBSTRUCTION OF THE BOWELS.

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THE affections which are here to be treated of present many features in common with enteritis, and their description is not infrequently included in the description of that disease. Actual enteritis does indeed occur at some period or another in the course of most of them; but their special claim to form a group by themselves consists in the fact of the existence in all of them of some mechanical impediment to the transmission of the contents of the bowels, in connection with which enteritis is apt to, but does not in all cases necessarily, supervene. They are: 1st, constipation; 2nd, stricture; 3rd, compression and traction of the bowel; 4th, internal strangulation; 5th, impaction of foreign bodies; and 6th, intussusception.

I. CONSTIPATION.

(a) *Pathology and Symptoms.*—Constipation not only forms a more or less essential element in the history of all the affections just enumerated, but of itself induces occasionally insuperable obstruction; and on both of these grounds demands some brief consideration here. Prolonged retention of fæces is, within certain limits, of such common occurrence, and is attended with so little inconvenience, that it scarcely deserves in a large number of cases to be regarded as an abnormal condition. It may doubtless be accepted as a general rule, that persons enjoying robust health, and unimpeded in the regular performance of their various functions, have an alvine evacuation at least once daily. Yet many who are apparently equally healthy have their bowels relieved habitually every two or three days only, or even but once in a week or fortnight. Cases indeed are not altogether rare in which some degree of good health has been maintained for many years although fæcal evacuations have during that time occurred only at intervals of six weeks or two months. In the case of a lady recorded by Dr. Robert Williams,[1] in whom habitual constipation appears to have been augmented by the constant use of large quantities of opium, the bowels were frequently confined for six weeks

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[1] Dr. Burne on Habitual Constipation, quoted by Mr. Pollock in Holmes's " System of Surgery," vol. iv.

together, and during one year of her life there were only four evacu-
tions at intervals of three months. It must not be forgotten, howev
that that degree of constipation which is habitual with one man, and i
him compatible with perfect health, may be and often is a source
discomfort, if not of positive illness, to another man in whom its occu
rence is exceptional. Thus, to most persons whose daily habits in th
respect are regular, the retention of fæces for two or three days is a
to produce not only local uneasiness, such as fulness, heat, tende
to piles and flatulence, but also some degree of general constitution
disturbance indicated by headache, foul breath, loss of appetite, a
dyspeptic symptoms, and not unfrequently terminates with more
less tenesmus, or even slight dysenteric diarrhœa. But even in ca
in which, from long habit, constipation has come to be regarded as t
normal condition of things, some of the above specified discomforts
actually for the most part coexist in some degree with it, but having b
come, like the constipation, habitual, cease to be observed, or at all ev
become tolerable. It is easy indeed to see that constipation must tend
produce various inconvenient results: the retention of a mass fro
which gaseous matters are being constantly evolved, is necessarily p
ductive of colicky pains and imperative desire to discharge flatus; t
constant pressure of a hard mass immediately above the anal out
causes not only congestion of the mucous membrane of the part, b
retardation of blood in the hæmorrhoidal veins, and ultimately piles,
produces also not unfrequently some degree of uneasiness in connect
with the genito-urinary organs; lastly, when defæcation occurs, the e
pulsion of the fæces is apt, in consequence of their bulk, and hardne
and dryness, not only to be attended with very considerable pain, a
perhaps some loss of blood, but to be followed by prolonged burni
or aching, and (as has been already pointed out) by more or l
dysenteric inflammation.

But much-prolonged constipation leads sometimes to other a
far more serious results, namely to dilatation and hypertrophy
the intestine, ulceration of its mucous surface, and perforation of i
walls with extravasation of fæcal matters into the peritoneal cavi
The dilatation is sometimes so great, that the colon measures fro
nine to ten or even twelve inches in circumference. It begins at
distance of one or two inches from the anus (which seems spa
modically contracted) and occupies more or less of the remaind
sometimes the whole length of the large intestine; in which latt
case the chief distension is observed in the rectum, sigmoid flexur
and cæcum. Hypertrophy of the muscular coat, which alway
accompanies dilatation, is general, but most marked in the sigmoi
flexure and upper part of the rectum, where the thickness may be
inch or more. When ulceration takes place, it is perhaps partly du
to yielding of the mucous membrane from over-distension, partly t
the constant irritation kept up by the fæcal mass within. Perfora-
tion may ensue, either while the constipation remains unrelieved,
and then either through the progress of ulceration or by laceration;

or after the bowel has been emptied, in consequence of the continuance of ulceration. Enormous quantities of fæcal matter are sometimes removed from patients suffering from aggravated constipation; in Dr. Williams's case above referred to, numerous round lumps, each the size of a large fœtal head, were passed at a time, and often in sufficient numbers to fill a common-sized pail.

I recollect two fatal cases which strikingly illustrate some of the observations which have just been made. The first was that of a little girl, eight years old, whom I saw casually only during life, and of whose history I obtained after her death some not very perfect details. She had long suffered from tendency to constipation; and it was stated that she had occasionally gone as long as three weeks without passing an evacuation. At the time of her admission into the hospital, there had been no relief of the bowels for seven weeks. She was then pale and thin, had a large tense belly, without pain or tenderness, a clean tongue, and a poor appetite. She had a "strumous" look, and was supposed, I believe, to be suffering from abdominal tubercle. She became gradually more and more emaciated and anxious-looking, while the belly grew larger and more tense. She never had any distinct abdominal tenderness, but suffered at times from colicky pains, and often (especially towards the close of life) complained that she was so full that she felt as if she should burst. During the last week or two the tongue became somewhat foul, and she had frequent vomiting, but never of stercoraceous matter. She passed but little urine, and that was high-coloured. She sunk gradually from exhaustion, and died exactly three weeks after admission. Amongst other kinds of treatment adopted was the use of purgative medicines and of purgative injections; and the medical man in attendance on her was led to believe that they had acted. There is no doubt, however, from subsequent inquiries, as well as from what was observed after death, that he was deceived. At the post-mortem examination, the form of the distended intestines was distinctly impressed on the tense and thin abdominal walls, and on opening the abdomen the enormously enlarged colon was at first alone visible. The distension began at the cæcum and extended to within two inches of the anus, where it ceased abruptly. In the greater part of its extent, the bowel measured from nine to ten and a half inches in circumference, the greatest amount of distension being manifested in the sigmoid flexure. The muscular walls were hypertrophied from the ascending colon to the lower end of the sigmoid flexure; and in the latter situation (where the hypertrophy was greatest) they measured $\frac{1}{8}$ inch in thickness. The mucous membrane seemed healthy in the greater part of its extent, but it presented some congestion here and there, and at distant intervals large patches in which there were groups of small circular shallow ulcers. The bowel contained no flatus, but was completely full of thick semi-solid olive-green-coloured fæces. These were more solid in the rectum than elsewhere, and immediately above the anus formed an indurated conical lump. The small intestines were also

considerably distended, though much less so than the larger bowel, and were filled throughout with semi-fluid olive-green-coloured contents. The stomach was small and healthy, and empty. There was no other disease. There can be no doubt that the death of the child was due to the neglect of simple constipation, that the indurated fæcal lump above the anal orifice had formed a plug which the bowel had been unable to expel, and which the accumulation of more and more fæces above and around it had served only to fix more securely. That the bowel had striven to expel its contents was shown by the hypertrophied condition of its muscular coat. A very similar case is recorded by Mr. Gay ;[1] but there the nature of the case was recognised, the rectum was relieved by mechanical means, and the child was saved. The second case referred to above was that of a young man, aged 24, who also had been the subject of habitual constipation ; and who on one occasion, after the persistence of constipation for an unusually long period, was attacked with diarrhœa, which lasted about six weeks, and was then followed by sudden peritonitis, of which he died. There was found after death inflammation of the peritoneum, due to a perforation in the transverse colon, great dilatation and thickening, yet almost complete emptiness of the whole length of the large intestine, and just the same kind of ulceration of the mucous membrane in patches as that described above. It was in one of these patches that perforation had taken place. Here, as in the former case, it is obvious that long-continued constipation had caused permanent thickening and dilatation of the large intestine, and ulceration of its mucous surface ; but here, additionally, after the relief of the constipation, the ulceration had provoked and maintained a condition of diarrhœa, and had ultimately caused perforation.

Constipation, in the sense in which the word is here employed, is probably always due to retention of fæces in the lower part of the large intestine, either from failure to respond to the desire for defæcation when the desire presents itself, or from sluggish action on the part of the lower bowel. It is very rare indeed, if there be no actual obstruction, that the contents of the alimentary canal do not pass along the whole length of the small intestine, and even along the colon, at a tolerably uniform rate ; at all events, any actual arrest of their transmission, unless it be owing to the presence amongst them of some massive foreign body, is probably never met with, except occasionally in the cæcum and sigmoid flexure.

Constipation is due to a variety of causes, and occurs under numerous different conditions, which it is scarcely necessary to enumerate here, far less to consider in detail. It is frequently caused temporarily by change of diet, scene, or habits, among which latter may be included anything which interferes with the regular performance of defæcation ; it happens commonly in various kinds of disease, and it occurs in a chronic form in chlorotic or dyspeptic girls and young women, and also in men and women (especially

[1] Path. Soc. Trans. vol. v.

the latter) of sedentary habits or of sluggish constitution. It occurs too, often perhaps as the result of habit, in persons, young and old, in whom no special cause for it can be recognised; and indeed, in many of the more remarkable cases that come under observation, it is quite impossible to assign a definite cause for it. Among local conditions which may be supposed to operate in a greater or less degree in the above cases, are : first, modifications in the character of the fæces such as we see in diabetes, where, owing to the rapid escape of fluid by the kidneys, they become preternaturally dry, and proportionately diminished in bulk; second, sluggishness on the part of the rectum ; and third, debility of the same part which may be primary, and due in the first instance to simple thinning and weakening of the muscular fibres, and which probably occurs virtually in all cases of long-continued constipation, when the bowel has becomes dilated, and on that account (even if the muscular coat be hypertrophied) less competent to contract efficiently on its contents.

(*b*) *The Treatment* of constipation must be made to depend more or less upon its cause, on its antecedents and on its effects. Where it is a mere temporary matter, depending on accidental circumstances, or arising in the course of acute diseases, its treatment is simple enough, and needs no description here. Where it has become a chronic affection, its causes should be investigated, and as far as possible obviated ; and it may be necessary to employ habitually mild aloetic or other purgatives, or enemata. Sometimes the application of galvanism to the surface of the abdomen, or to the abdomen and anus, is efficacious. But iron and other tonics also are frequently of advantage ; and strychnia is by many believed to be of great value. In cases in which the rectum becomes filled with a hard immoveable mass, and the bowel above distended in consequence with accumulated contents, the evacuation of the rectum by mechanical means becomes essential. This may be effected sometimes by the use of the finger or of a spoon, or some such instrument ; sometimes by the employment of copious enemata administered in the ordinary way ; or, better still (as in Mr. Gay's case), by directing a forcible stream of warm water, conducted from a height by means of a tube, into the rectum, allowing it to play upon the fæcal mass for half an hour or so at a time, and thus to cause its disintegration and either effect or facilitate its removal.

II. STRICTURE.

By this term is meant a circumscribed diminution in the calibre of the bowel, due either to contraction of the mucous and sub-mucous tissues (the consequence usually of ulceration), or to some deposit or growth involving the general thickness of the walls and encroaching on the canal, or to some spasmodic action of the circular muscular fibres. It is occasionally the result of malformation.

(*a*) *Pathology.*—Congenital stricture, though in some of its forms by no means rare, is an affection the treatment of which belongs almost exclusively to the surgeon, and one, therefore, that needs little more than incidental mention here. It is limited, indeed, with few exceptions, to the lower extremity of the bowel—the rectum and the anus—one or both of which parts may be found at birth to be impervious or absent, or reduced to a mere fistulous canal or orifice, while, in addition, the lower end of the fully-dilated bowel above occasionally communicates with the vagina in the female, or with the bladder or urethra in the male. Very much more rarely, congenital stricture is met with in the duodenum, at or above the point at which the common bile duct discharges itself. Two cases of this kind are recorded in the twelfth volume of the "Pathological Society's Transactions," one by Dr. Wilks, the other by Dr. G. Buchanan. In both, a kind of membranous septum existed at the point referred to, and the portion of duodenum above was thickened and dilated, forming a mere prolongation of the pyloric end of the stomach. In Dr. Wilks's case the bile duct opened immediately below the septum, which was impervious; and the child died at the end of thirty-eight hours, its death being preceded by vomiting and convulsions. In Dr. Buchanan's case, the duct opened on the under-surface of the septum, the septum presented a minute central orifice, and the child, a girl, lived eighteen months. According to the history, she was apparently quite well up to within a month of her death, probably because (as is supposed) she had hitherto been fed only from the breast and with milk. She appears during the last month of life to have suffered from constant vomiting, great restlessness and uneasiness or pain, together with (during the earlier part of that time) frequent convulsions. It may be added, that in this case, where the parts were examined with much minuteness, the septum was ascertained to consist of a duplicature of mucous membrane, not unlike an enlarged valvula connivens, enclosing a few scattered muscular fibres prolonged from a stout circular band which surrounded its base.

Although spasm of the circular muscular fibres has been given above as one of the causes of intestinal stricture, and although it doubtless does form a very important element in many cases of fatal obstruction of the bowels, it is certainly of very rare occurrence, as an independent affection, and may be considered practically as limited to the rectum and anus. And indeed, even in these parts, spasmodic obstruction is probably always attended with some ulceration of the adjacent mucous membrane, to which there is reason to believe it secondary. Thus spasmodic contraction of the sphincter ani, an affection which may be regarded as exclusively surgical, seems to be dependent on the formation of an ulcer, at or within the verge of the anus; and not very infrequently spasmodic contraction, with great hypertrophy of the muscular tissue, is met with as one of the troublesome sequelæ of dysenteric ulceration of the rectum.

But the varieties of stricture with which we have here to deal particularly are those in which, according to the definition with which we started, the stricture is due either to the contraction of the mucous and submucous tissues, or to some deposit or growth involving the general thickness of the walls. The cicatrization which follows ulcerative destruction of the mucous membrane is a common cause of diminution of the calibre of the bowel. But what particular kinds of ulceration are most apt to be followed by this condition is not very clear. Indeed, in most cases where stricture from ulceration is found after death, there is nothing in the history to guide our judgement in this respect. It is certain, however, that in order to produce any marked constriction, the area of ulceration must either have been considerable, or must have extended round the bowel. There is reason to believe that irritant poisons, in consequence of their corrosive effects on the mucous membrane, lead occasionally to the production of stricture of the intestine, especially in its upper part, just as they occasionally cause œsophageal stricture. There is no doubt that tubercular ulceration of the bowels, which very commonly forms annular patches or occupies extensive tracts, and which not at all infrequently undergoes more or less perfect cicatrization, is a yet more frequent cause of stricture, either in the lower part of the ileum, or in the cæcum, or in some part of the colon. Dysenteric ulceration of the large intestine is also a distinct cause of stricture; as again is the separation by sloughing of an invaginated portion of bowel. The ulcers of typhoid fever, on the other hand, are known to result very rarely, if ever, in obvious contraction of the calibre of the bowel: although it is pretty certain that even in this case, when the ulceration has spread and become extensive, marked constriction may attend its cicatrization. When stricture is due to ulceration, we find the mucous surface contracted, sometimes completely cicatrized, sometimes presenting unhealed spots of ulceration, with fungous excrescence or granulations, and separated from the subjacent muscular coat by a more or less abundant deposit of dense fibroid tissue. The stricture itself may be a mere ring, or it may occupy several inches of the length of the bowel; I have seen the whole cæcum thus reduced into a channel barely capable of admitting a goose's quill. Another cause of stricture, limited probably to the large intestine, is the growth of that fibroid material which resembles, but has of late been distinguished from, true scirrhus. This generally involves all the coats to a greater or less extent, encroaching, as it grows, upon the intestinal tube. Sometimes, but not necessarily, its surface ulcerates. A growth probably identical with this, occurring in so-called "pelvic cellulitis," sometimes involves the walls of the rectum and causes stricture there. But by far the most frequent cause of stricture is the development of cancerous disease in the coats of the intestine. This is sometimes local, or at all events of primary origin in the bowel, being then, perhaps without exception, a disease of the large intestine; but more frequently it involves the gut by spreading to it from some neigh-

bouring part, as from the peritoneum, the mesenteric or other abdominal lymphatic glands, from the substance of the gastro-hepatic omentum, from the cellular tissue of the venter ilei or pelvis, or from the genito-urinary organs.

The presence of a stricture is always a more or less serious impediment to the progress of fæcal matters along the bowel; and in all cases therefore leads in a greater or less degree to certain results. These are: first, undue accumulation of fæcal matter above the stricture, with proportionate dilatation of the bowel there; second, hypertrophy of the muscular parietes of the dilated bowel; and third, diminution in calibre and even atrophy of the bowel below. It is an interesting fact that, in cases of stricture of the colon, the greatest degree of dilatation is often found, not in the portion of intestine immediately above the stricture, but in the cæcum. The tighter and the longer a stricture, the more exaggerated, other things being equal, will be the several consequences just described; and the more danger will there be of the supervention of permanent obstruction. Yet it is a very remarkable fact, that very tight strictures are not infrequently found after death in cases in which during life there has been no suspicion of their presence. Allusion has been already made to a case which was under my own care, wherein the cæcum was contracted into a channel two inches long, and about the size of a goose's quill; yet the patient had no symptoms of stricture, and died of acute pneumonia. But it is in the small intestine especially that stricture is apt to be present without producing any of its characteristic symptoms—a phenomenon which is probably due, in part at least, to the fact that the contents of the small intestine are usually much more fluid than those of the large, and are consequently much more readily propelled through a [very narrow orifice. Indeed, Dr. Buchanan's case already cited, and many others that might be quoted, show clearly, what also common sense would lead us to surmise, that the more solid the matters are which ought to be forced through a stricture, the more likely are they to be arrested there, and thus to render the obstruction complete. It may be added, that the lodgment of fæces above a stricture is very apt, not only to prevent the complete cicatrization of the ulcer by which the stricture itself may have been originally produced, but to cause erosion and ulceration in the dilated bowel above, a contingency which is still more likely to arise when cherry-stones or plum-stones or other hard bodies form a part of the accumulation. And, further, it may be added, that perforation of the bowel at or above the seat of stricture is not of very infrequent occurrence, generally as the result of perforating ulcer, occasionally as the result of laceration from associated softening and over-distension.

Stricture may be met with in any part of the intestine, yet it occurs in different parts with very different degrees of frequency. The published statistics of fatal cases show that its occurrence as a fatal disease in the small intestine is comparatively rare (according to Dr.

Brinton,[1] in 8 out of every 100 cases), and that as regards the large intestine (to quote again Dr. Brinton's figures, with which those of other writers agree pretty closely), out of 100 fatal cases, 4 are in the cæcum, 10 in the ascending colon, 11 in the transverse colon, 14 in the descending colon, 30 in the sigmoid flexure, and 30 in the rectum. Dr. Brinton calculates that stricture occurs three times in men to twice in women; and that the average age at death is $44\frac{2}{3}$ years.

(b) *The Symptoms* to which stricture gives rise vary greatly according to circumstances, especially according to its position, its degree, its cause, and its complications. As has been already pointed out, stricture of the small intestine very rarely causes symptoms sufficiently characteristic to enable us to diagnose its presence, and rarely causes death except by the accession of complications which themselves are not distinctive. It probably gives a liability to colicky pains, and to some degree of nausea and sickness. Indeed, in the case of the large intestine the symptoms produced by stricture may be for a long time vague and inconclusive, and even misleading. The patient suffers perhaps for weeks, or months, or years, with occasional attacks of colicky pain, associated, it may be, with more or less constipation; but not infrequently during the earlier period of his malady diarrhœa may be a yet more prominent symptom. If, however, the obstruction be in the vicinity of the rectum, solid motions generally soon assume a narrow tape-like or pipe-like form. Occasionally the symptoms of obstruction come on quite suddenly; but most frequently some degree of constipation long precedes the occurrence of complete obstruction; and sometimes, too, it happens that the patient, previous to his final attack, may have experienced one or two or more similar attacks, which have, however, yielded to treatment. The symptoms which attend and indicate impassable stricture are insuperable constipation, painful peristalsis coming on periodically, and often rendering itself audible by borborygmi and visible through the abdominal walls, abdominal fulness and uneasiness, followed after a time by nausea and vomiting—the vomited matters becoming finally stercoraceous—and death at last from simple asthenia. Febrile symptoms and abdominal tenderness are often absent from first to last: but sometimes inflammation supervenes, or perforation takes place, and then enteritic or peritonitic symptoms become superadded. When the case is free from these or other complications, its progress is essentially chronic, and the patient, if not improperly treated, lives for a considerable time, often for many weeks. The duration of life in these cases may be said somewhat roughly to vary between two weeks and three months. Indeed, when we consider that constipation may continue for three months or more with comparatively little injury to the system, it is impossible not to believe that persons with simple impassable stricture of the rectum may, under favourable circumstances, survive for even a longer period than that.

[1] "Intestinal Obstruction," by William Brinton, M.D. F.R.S. 1867. Frequent reference is made to this work throughout the present article.

It is always satisfactory, and sometimes highly important, to ascertain the exact site of stricture ; and in coming to a conclusion on this point, it is well to bear in mind that at least three-fourths of the strictures of the large intestine are situated to the left of the mesial line of the abdomen. We need not, however, in all cases limit ourselves to a simple calculation of chances. It is natural to believe that the distension of the bowel above the stricture, and its collapse below, should reveal themselves to manual if not to ocular examination of the abdomen, and sometimes, no doubt, the form and position of a struggling, or even of a quiescent, length of distended bowel, may by such means be clearly identified. Fulness and dulness and weight in the course of the cæcum and ascending colon, or on the right side of the belly, might thus indicate a stricture at or about the hepatic flexure, and, associated with the same conditions extending across the epigastrium, might indicate stricture at the splenic flexure or in the descending colon ; whereas fulness, and the like, limited to the left side of the belly, or most pronounced in that region, might equally be indicative of stricture in the sigmoid flexure or rectum. But thickness, or rigidity of the abdominal walls, or tenderness, or the presence of tumours, or the altered positions which greatly distended tracts of bowel are apt to assume, often interfere to prevent the easy recognition of even extreme differences of intestinal dilatation and fulness. Dr. Brinton maintains that the amount of fluid which may with care be injected *per anum*, is a very valuable guide in estimating the point of stricture. He says : " With a maximum injection of a pint of warm bland liquid, the obstruction of an ordinary male adult may be referred to a point not lower than the upper end of the rectum. A pint and a half, two pints, three pints, belong to corresponding segments of the sigmoid flexure. The descending and transverse colon accept a larger but more irregular quantity." But here again there is evidently very abundant room for error ; for it is certain that not all contracted bowels are tolerant in an equal degree of mechanical distension, and there can be no doubt that a stricture, which may prevent the passage of hard fæcal matter in one direction, may yet allow of the transmission of thin fluids in the opposite direction. Lastly, when the stricture is a short distance only from the anus, its presence may often be ascertained by the introduction of the finger, or, as has been suggested, of the entire hand ; and if it be beyond the reach of actual touch, yet in the rectum, the careful introduction of a bougie may perhaps reveal its position. But it must not be forgotten that the curvatures of the rectum, and the prominent folds of its mucous membrane, are such impediments to this latter mode of examination as to rob it of very much of its value ; in addition to which, it is attended with, at all events in many cases, considerable risk of damage.

(*c*) *Treatment.*—Whenever we have reason to believe in the presence of a stricture, it is obviously desirable that nothing which is not in a perfectly fluid or pultaceous condition should be allowed to enter the bowel,—therefore, that the food taken habitually should be easy of

digestion, thoroughly well masticated, and not more abundant than is absolutely necessary for the preservation of health, and especially that neither plum nor cherry stones, nor even pips, should be swallowed; secondly, that the bowels should themselves be kept as far as possible in a quiet condition,—in other words, whilst constipation should as far as possible be prevented, diarrhœa and painful gripings should equally be guarded against. If there be constipation, it may be directly relieved, or the bowel above the seat of stricture may be encouraged, as it were, to propel its contents by the use of simple non-purgative enemata; but purgatives of all kinds, certainly anything like active purgation, should be religiously eschewed. Should the stricture be in the rectum, and within reach, it may of course admit of dilatation and relief by the use of a bougie. When symptoms indicative of complete stoppage manifest themselves, the wish to employ active measures to relieve the patient's distress naturally obtrudes itself; but such measures are for the most part even less admissible now than formerly. Enemata may be of advantage, partly, as before pointed out, to guide our judgment as to the seat of the stricture, partly (if the stricture be in the large intestine) for the purpose of promoting the relief of the bowel above the impediment; but purgatives are not only useless, but almost certain to do serious mischief, if not to cause actual perforation. On opium and other sedatives, and soothing applications locally applied, utterly inadequate though they generally are, must yet be our chief reliance, so far as ordinary medical treatment is concerned. But in all such cases a time comes when the advisability of forming a communication from without with the portion of bowel above the stricture—in other words, the attempt to establish an artificial anus—becomes a serious question. When the stricture is in the large intestine, as it generally is, Amussat's operation, in one or other loin, is that which would of course be chosen for performance; and although it is obviously incompetent to cure the stricture, it avails very often to prolong life, and sometimes to prolong it for a considerable period. If the stricture happens to be in the small intestine, Litré's operation is alone available.

III. Compression and Traction.

Dr. Hilton Fagge[1] has with great reason distinguished on the one hand from stricture, on the other from internal strangulation, a class of cases related to both, which is yet clearly distinguishable from them, and which he designates "Contractions." They are cases in which the bowel becomes obstructed by the compression, or the pressure, or the traction exerted upon it by adhesions, or growths, or deposits, situated externally to it, and in which there is no contraction inherent in the walls themselves, and not necessarily or generally any strangulation.

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[1] In an excellent paper in the Guy's Hospital Reports for 1869, to which frequent reference is made in the course of this article.

(a) *Pathology.*—Under the above heading may be included those cases in which the rectum becomes obstructed, and defæcation rendered painful or difficult, in consequence of the pressure exerted on that part of the bowel, either by an enlarged or displaced uterus, or by a uterine or ovarian tumour. It is conceivable, of course, that any form of abdominal tumour may by pressure obstruct the alimentary canal in some part of its course. I recollect one case of death by rupture of the abdominal aorta, in which the blood, effused and coagulated in the sub-peritoneal tissue, had so surrounded and compressed the third part of the duodenum, that the finger passed along it with difficulty; and while the stomach and duodenum above contained a considerable quantity of contents, the intestine below was perfectly empty.

But the cases which are now more particularly referred to are those in which obstruction is due to the embarrassment of a greater or less length of bowel, caused by the presence on its outer surface of lymph or false membrane, which binds it more or less firmly to the surrounding parts, and sometimes constricts, sometimes leads to the formation of sharp angular bends. The adhesions are often produced by circumscribed peritonitis, but more frequently, perhaps, are developed in the course of peritoneal tubercle or cancer. In some cases the intestine has been incarcerated in a hernia, and portions of it have become invested in adhesions, which attach it, perhaps, to the neck or some other part of the sac, or to the omentum; in others, the transverse colon or sigmoid flexure, or some other tract of bowel, is hooked down, as it were, by bands of lymph to the uterus, or ovary, or some other structure within the pelvis; in others, again, several contiguous coils of small intestine are tightly bound together, forming a kind of tangled mass. Fatal cases of compression or traction always furnish distinct evidence of more or less complete obstruction, in the contraction and emptiness of the bowel below, and in the dilatation, hypertrophy, and fulness of the bowel above; but the part in which the actual obstruction has taken place, though contracted and more or less empty, is frequently found to admit with ease of the passage of the finger, or even of some larger body. The immediate cause of obstruction indeed is not generally a simple tight constriction, but consists sometimes in a comparatively slight compression of a considerable length of bowel, which thus becomes embarrassed in its action, and sometimes in the presence of a sudden bend or twist, the upper portion of which becoming distended presses upon and flattens the portion beyond, and so renders it impervious, and in association with these doubtless a greater or less degree of spasmodic contraction. Sometimes, however, the obstruction is as sharp and definite as any stricture.

Dr. Fagge points out (and in the opinion which he expresses I entirely agree with him) that these cases are of far more frequent occurrence in the small intestine than in the large, and that in a clinical point of view they may be regarded as the strictures of the smaller bowel.

b) Symptoms and Treatment.—The symptoms of the affection now
der consideration are almost, if not quite, identical with those of
cture. In both cases, when the impediment to the due action of the
vel is associated with abdominal cancer or tubercle, or any other form
adventitious growth, the symptoms connected with these complica-
as mask, if they do not conceal, the symptoms due to obstruction.
both cases, when no such complications are present, the symptoms
netimes come on quite suddenly, sometimes creep on insidiously with
asional colicky pains, limited but powerful peristaltic movements,
l gradually increasing obstinacy of the bowels; and sometimes the
ient suffers from one or more severe attacks of total constipation,
ich yield after a time to nature or to treatment, and in this respect
y differ from the final and fatal attack. In both cases, again, the
ease, though not entirely free from the danger of the supervention
peritonitis or enteritis, is still not necessarily complicated with
nptoms of inflammation, and its course, therefore, tends to be
uliarly chronic, lasting sometimes for weeks, and its close is
ually determined by gradual exhaustion only. Dr. Fagge thinks
t cases of this kind are to be distinguished by their chronicity,
the occurrence of obstruction rather in the small intestine than
the large, and by the powerful and well-marked vermicular
vements which occur, often nearly to the last, in the length of
vel above the impediment. He points out that it is in cases of
onic impediment especially that the bowel above becomes hyper-
phied as well as dilated, and he argues that it is therefore probably
these same cases (stricture and compression) that the movements
the bowel, in their endeavours to overcome the impediment, are
st powerful and most obvious. In confirmation of this view, I may
te that the cases in which I have myself most distinctly traced the
istaltic movement of the bowel have been cases of the kind in
estion.

[t is needless to draw any distinction here as regards treatment
ween stricture and compression of the bowel.

The following case may be quoted as a typical example of the
ction which has just been described. A man, forty years of age,
s attacked suddenly, seven weeks before his admission into St.
omas's, with severe colicky pains, which confined him to his bed
two or three days. He improved, but at the end of a few days
l a recurrence of the same symptoms, lasting for about three
eks, and attended with nausea, vomiting, and constipation. Then for
days he became free from pain and apparently convalescent. But
days before his admission all his symptoms returned with in-
ased severity; and during this time vomiting was pretty constant
l his bowels remained unopened, although strong purgatives were
eral times administered. On admission his face was anxious, but
tongue was clean and his pulse quiet. He vomited regularly two
urs after taking food. The belly was distended and tympanitic,

and somewhat tender; he complained of constant pain in it; and severe exacerbations of pain, lasting two or three minutes and attended with a gurgling sound, came on about every five minutes. The vomiting became stercoraceous four days after admission, and continued so thenceforth. The bowels were never acted on except by enemata, which brought away fæcal matters in gradually decreasing quantities. The distension and tenderness of the belly continued, if they did not increase; and the paroxysms of more intense pain coming on every few minutes troubled him almost to the last. During these paroxysms, the violent peristaltic movements of the bowels could be followed through the abdominal parietes with the greatest facility. He had no distinct febrile symptoms, and no hiccough; he continued perfectly sensible, and died of simple exhaustion just three weeks after admission. At the post-mortem examination, the small intestines generally were found to be enormously distended, and their surface a little heightened in colour, and marked with longitudinal bands of rather intense capillary congestion. From the middle of the ileum to within a foot of the cæcum the coils were adherent to one another and to the brim of the pelvis by bands and filaments of false membrane, and were so entangled that their direction was traceable with difficulty. The portion of bowel involved was for the most part somewhat dilated; its lowest third, however, was contracted and empty, as also was the portion between this and the cæcum. The stomach and small intestines down to the seat of contraction were dilated and full of thin pea-soup-like fluid; the cæcum and large intestine were contracted throughout, but here and there in the ascending colon were small lumps of hardened fæces. The mucous membrane of the alimentary canal was healthy everywhere. There was no hernia, no intussusception, and no part of the bowel along which the finger could not readily be passed.

IV. INTERNAL STRANGULATION.

Internal Strangulation arises from similar causes to those which produce ordinary strangulated hernia, namely, constriction or nipping of a portion of bowel by the edges of some natural or artificial orifice through which it protrudes, with consequent arrest of the circulation of blood in it, and impediment to the passage of fæcal matters along it. Such orifices are the foramen of Winslow, congenital or acquired perforations in the mesentery, meso-colon, great omentum, or other peritoneal duplicatures, or apertures formed, with the aid of neighbouring parts, by bands of fibroid tissue (the result generally of some inflammatory process) extending from one point of the peritoneal surface to another. And it is obvious that the same accidental conditions which lead to the protrusion of intestine into an ordinary hernial sac, may equally lead to the protrusion of a knuckle or loop or still larger mass of bowels into one of these. But, of course, if

o more follows in the one case than in the other that strangulation
hould either immediately, or at any subsequent period, follow upon
his displacement; although in both cases there is always imminent
anger of its occurrence.

(*a*) *Pathology.*—Protrusion of bowels through the foramen of
Winslow must be an exceedingly rare event. Rokitansky,[1] however,
lludes to a case in which he found this the cause of strangulation
f a large portion of small intestine. Perforation of the various
uplicatures of peritoneum, with the passage of intestine through the
erforation, and consequent strangulation, is of much more frequent
ccurrence. This accident appears to be most common in con-
exion with the mesentery, and then generally to follow upon lacera-
on from violence. Next probably in order of frequency it is met
ith in connexion with the great omentum. And cases are recorded
which death has followed the strangulation of a portion of bowel
rough a hole in the duplicature of peritoneum belonging to the
ermiform appendix, or through a hole in the suspensory ligament
f the liver, or in the broad ligament of the uterus. Meso-colic
pture is probably a congenital malformation. Three cases of it are
corded in the "Transactions of the Pathological Society;" and in
ach of them nearly the whole mass of small intestines was con-
ained in a large pouch of the transverse meso-colon, or in the
esentery of the transverse and descending colon. In two of them
eath was due to disease independent of the rupture; in the
ird, recorded by Dr. Peacock, the patient died of strangulation.
here is probably no part of the peritoneal surface to which bands
apable of producing strangulation may not be attached; but there
re certain structures and certain conditions of parts with which they
re specially apt to be connected. Thus, the vermiform appendix
ften becomes adherent to neighbouring structures, such as the
esentery, small intestine, colon and ovary, forming a kind of loop;
hus, too, diverticula of the lower extremity of the ileum become
ttached, with a similar result, usually by the apex, either to the
esentery or some other neighbouring part, or are prolonged to the
mbilicus in the form of a cord (a remnant of fœtal life). Again,
ands producing strangulation are often joined to the mesentery, or
he parts concerned in old ruptures; and often to the pelvic organs,
ore particularly the uterus, Fallopian tubes, and ovaries. It may
ere be noted also that strangulation is not very infrequently pro-
uced by the slipping of a loop of intestine under the lower edge of
he mesentery (unusually elongated), of a portion of bowel hanging
ow into the pelvis, or even under the pedicle of an ovarian or uterine
umour. Finally, there are rare cases of internal strangulation, in
hich the bowel protrudes into a lacerated bladder or uterus, or
nto a perforated bowel, or through the diaphragm. Cases also are
ccasionally met with in which there is a free communication, gene-

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[1] Pathological Anatomy : Sydenham Society's Translation, vol. ii.

rally, if not always congenital, between the peritoneum and pericardium, or one of the pleuræ.

The small intestine is much more frequently strangulated than the large; and of the large intestine the regions most liable to this accident are those which are most moveable, namely, the cæcum and sigmoid flexure. Internal strangulation occurs at any age; generally, however, above thirty; but strangulation in connexion with the appendix vermiformis or a diverticulum happens most frequently in comparatively early life, the average age being, according to Dr. Brinton, twenty-two years; further, strangulation from diverticula and from lacerated mesentery is, according to all authorities, far more common among males than females. It has already been pointed out that there is a very important relation between peritoneal bands and the sacs of old herniæ, and in females between such bands and the pelvic organs.

(b) *Symptoms.* — The symptoms of internal strangulation are identical with those of ordinary strangulated hernia, and so like those which have been described as the symptoms of the severer form of enteritis that there is no occasion to give here any special account of them. It need scarcely be added that they differ essentially from those of stricture and of compression of the bowel, in the facts that they are always sudden in their origin and acute in their severity and progress, and always end fatally (if the stricture be not relieved) within a few, rarely more than five or six, days.

(c) *Treatment.*—As regards the general management and medical treatment of these cases, nothing can be added to what has already been laid down in reference to enteritis. But whenever the diagnosis of an internal strangulation has been made, it must of necessity become a question whether an operation should be performed with the object of relieving it. There can be no doubt, of course, that the liberation of a portion of bowel strangulated by any of the various causes above enumerated ought *cæteris paribus* to be attended with as good results as the division of the stricture in ordinary cases of strangulated hernia; but there is also no doubt that operations performed with that intention have not on the whole afforded any encouraging results. When, however, we consider that although typical cases of the different kinds of intestinal obstruction may really present characteristic peculiarities of symptoms, it is yet for the most part exceedingly difficult in practice to discriminate the cases that come before us, and that therefore operations must comparatively often be performed where from the nature of things they must be useless; and, further, that while even in the case of the operation for ordinary strangulated hernia its early performance is generally essential for its success, in the case of internal strangulation the operation, if performed at all, is almost always delayed until a late stage in the disease; it is not hard to understand why so little success has attended the operative treatment of the cases under consideration. A sufficient number of operations has, however, been successful

to justify us in laying it down as a rule, first, that in every case in which we have come to the conclusion that a patient is suffering from internal strangulation, an operation should be performed for its relief ; second, that in all cases in which we think it not improbable that such a strangulation exists, the patient should not be allowed to die without an exploratory operation having been effected or at least proposed.

(*d*) *Note on Torsion or Twisting of Bowel.*—There is a class of cases, far from uncommon, which may be conveniently adverted to here. They are cases of what is called " Torsion " or " Twisting " of the bowel. It has already been shown that fatal obstruction to the passage of fæcal matters along the bowel may be caused, or appear to be caused, by the formation of some abnormal abrupt bend, or twist, in connexion usually with external adhesions. In these cases, however, death is caused, as in stricture or compression from without (with which last I have classed them), by obstruction alone. But in the cases now to be considered, the twisted portion of bowel becomes the seat of enteritis, and death results speedily, with the symptoms of enteritis rather than those of obstruction. The cases, indeed, clinically seem to be undistinguishable from cases of enteritis or internal strangulation. The onset of the disease is sudden, the symptoms acute and severe, and the supervention of collapse and death speedy. And on examination after death there is found a length of bowel greatly dilated and black with congestion and inflammation, if not gangrene, no strangulation, at least no strangulation in the ordinary sense of the word, but instead, a remarkable twisting of the inflamed tract of bowel with its mesentery, by which twisting it is supposed that the vessels leading to and from the part have become obstructed. Such twisting, associated with inflammatory mischief, is sometimes observed in the small intestine ; but it is far more commonly met with in connexion with the larger bowel, and especially with the sigmoid flexure and cæcum. If these cases be really, as is generally believed, cases in which strangulation of the bowel is produced by the twisting of itself and its mesentery, they naturally fall under the head of internal strangulation, with which, as has been pointed out, their symptoms and progress ally them. I must confess, however, that I have a strong inclination to believe that most, if not all, recorded cases of this affection are essentially cases of enteritis, and that the twisting is a secondary phenomenon only. It is not very easy to see how a portion of bowel, unless its position be altered and its movements interfered with by adhesions (and certainly in many of the cases no adhesions whatever are observed), can become so twisted by any movements of its own, or even by the pressure of surrounding healthy parts, as to be either strangulated or incapable in virtue of its own peristaltic movements of recovering its normal position ; but it is easy to see that an inflamed and paralysed portion of intestine, heavy with accumulated contents, dilated to many times its normal

bulk, and forming a doughy, inelastic, inert mass, may under certain conditions by its mere weight subside from its normal site, or be pushed aside by the pressure of the actively vital parts around it, and so be made to assume a position and form suggesting the generally received explanation of the sequence of events.

V. IMPACTION OF FOREIGN BODIES.

It has already been pointed out that mere ordinary intestinal contents, no matter how unwholesome, how indigestible, or how imperfectly comminuted the ingesta from which they are derived may be, very rarely indeed cause by their accumulation permanent intestinal obstruction ; yet it is not improbable that, according to the ordinary belief, undigested masses of food do sometimes, in their passage along the small intestine, move with difficulty, or become temporarily impacted, and so produce pain and sickness, and even symptoms of obstruction. Dr. Brinton describes a case of this kind, in which he asserts that he distinctly traced by palpation a mass of half-chewed filberts in its passage (lasting two days) along the small intestine.

(*a*) *Pathology.*—Foreign bodies, indeed, of comparatively small size, such as coins, fragments of bone, teeth, marbles, plum-stones and cherry-stones, generally pass along the healthy intestine without causing any material inconvenience; and occasionally even pointed bodies—pins and the like—prove equally innocuous. They are all, however, a source of serious danger in the presence of strictures, above which they usually become arrested, or in which they may become lodged. The smaller ones among them may lead also to serious results by slipping into a diverticulum, or into the vermiform appendix; and those which are pointed are apt to perforate the intestinal wall, and thus, escaping into the peritoneal cavity, to set up fatal peritonitis, or, escaping into the surrounding tissues, to provoke suppuration there. In the latter case, the foreign body sometimes emerges through the abdominal parietes, sometimes (when it perforates the rectum) is the cause of anal fistula.

Insoluble matters, in the form of powder, or in a fibrous state, which under ordinary conditions may be swallowed with perfect impunity, occasionally, after having been taken habitually in large quantities and for long periods, are found to have been gradually deposited from the fæcal contents of the bowels, and to have concreted into hard masses. These are sometimes round or ovoid, and may then be termed intestinal calculi, and sometimes form casts of the portion of gut in which they lie. The former are probably always found in the large intestine; the latter rarely, if ever, occupy any other position than the rectum. Among substances which thus occasionally form concretions, are sesquioxide of iron, carbonate of magnesia, insufficiently cooked starch, and oat-hair derived from oat-cake and other articles of food made from oats.

Amongst cases of exceptional rarity may be included those which are here and there recorded of persons who have been in the habit of swallowing knives, or pins, or string, or hair, or cocoa-nut fibres; things which, from various causes, are somewhat difficult of transmission, and which with the constant additions which are made to them gradually form accumulations or masses, which sometimes attain very considerable dimensions, and may then easily be distinguished through the abdominal walls. These are generally found to occupy more particularly the stomach and upper part of the small intestine, and, when composed of fibrous substances, take the shape of the cavity in which they have formed. Their presence causes gradual dilatation of the part in which they are lodged, then congestion, inflammation, and ulceration, and finally, either perforation into the peritoneal cavity, or complete obstruction. It is remarkable, however, how long a period often elapses before such cases terminate in death, and how little, comparatively, of distress or even inconvenience the patient often experiences previous to the supervention of fatal symptoms.

But the usual cause of fatal impaction, and that which comes more especially within the scope of the present article, is the escape of a large gall-stone from the gall-bladder into the small intestine. The gall-stones here referred to are not those which so commonly slip from the gall-bladder into the cystic duct, and thence into the common duct, and thence (if they do not become firmly fixed there) into the duodenum : for although these cause grave symptoms enough so long as they are retained within the biliary passages, they cease, as a rule, to cause any ill effects so soon as they have gained an entrance into the bowel; their comparative smallness allowing them to pass along the intestines and to escape with the fæces, just as a plum-stone or a cherry-stone might do. The biliary concretions which become impacted in the bowel are single stones, or masses of coherent stones, of considerable bulk, varying, at a rough estimate, from three to four inches in circumference, and from one inch to two, three, or even four inches in length; in the former case presenting more or less of the ordinary cuboidal form, in the latter case forming a more or less complete cast of the gall-bladder. It is obviously scarcely possible that concretions of this magnitude can escape from the gall-bladder *per vias naturales;* and there is reason to believe that in all cases where a careful examination has been made, an ulcerated opening has been discovered, by which the cavity of the gall-bladder and that of the duodenum were in tolerably free communication, and through which the concretion had obviously escaped from its bed. When a large calculus has thus got into the duodenum, it seems to be carried on with the other contents of the bowel by means of the ordinary peristaltic movements. But its mere bulk prevents it from moving readily : besides which it provokes by its shape and hardness, as well as by its bulk, some irritation, if not inflammation, of the mucous surface over which it passes, and more

or less spasmodic contraction of the muscular tissue which surrounds it. It hence continues to progress irregularly, now moving slowly, now coming to a standstill, impelled onwards by the *vis à tergo*, checked in its passage by the spasmodic contraction of the portion of bowel which embraces it, and by the comparatively empty and contracted state of that which is below it, and causing as it descends more and more mischief to the mucous surface, until finally it becomes impacted, sometimes in the jejunum, sometimes in the ileum, and not unfrequently just above the ileo-cæcal valve. Then all the effects of complete obstruction, conjoined with those of intense enteritis, supervene ; the bowel below becomes empty, that above distended with accumulated contents, and generally more or less inflamed, while at the seat of obstruction and in its immediate neighbourhood the inflammation becomes intense, extending speedily to the peritoneal surface, and ends not rarely in gangrene and in perforation. Gall-stones rarely, if ever, become lodged in the cæcum, colon, or any other part of the large intestine.

Gall-stones are a product of the later period of life ; and hence obstruction by gall-stones can only be looked for at an advanced age. It occurs indeed rarely before the fiftieth year, and, it may be added, much more frequently in women than in men. Dr. Brinton estimates the average age of its occurrence at $53\frac{1}{3}$ years, and that it occurs four times as frequently· in women as in the opposite sex.

(*b*) *Symptoms and Treatment.*—The symptoms which indicate obstruction of the bowels by a gall-stone are as nearly as possible identical with those which attend internal strangulation or enteritis. The cases themselves are, however, amongst the most violent in their symptoms and the most rapid in their course of all cases of intestinal obstruction ; conditions which result partly from the intensity of the inflammation which attends them, partly from the fact that the obstruction is almost without exception situated in the small intestine, and often high up in it. Dr. Brinton calculates their average duration at five days. There are two or three circumstances which may afford more or less assistance in the discrimination of obstructions by gall-stones : such are, first, the age and sex of the person attacked ; second, the possibility in certain cases of discovering by palpation the presence of a gall-stone (that is to say, of a solid mass) in the bowels, and even of tracing in some degree its progress; and third, the occurrence of precursory symptoms due to the escape of the gall-stone from the gall-bladder, and to its presence in the bowel in the interval between this escape and its final impaction. It must not be forgotten, however, that in practice not only do we often fail in these cases to recognise a lump, or to obtain a history of premonitory symptoms ; but that we may have both a lump and a history in cases where the symptoms are wholly independent of the presence of a biliary calculus or other foreign body. There does not appear to have been observed any connexion between ordinary "attacks of gall-stones," and the affection now under consideration.

This circumstance, however, is not remarkable, when it is borne in mind that gall-stones which escape by the normal route must necessarily be small, and that the escape of one such stone makes the way of escape for others that may be in the bladder compara- tively easy, whereas those which cause intestinal obstructions are always large, and are often casts of the gall-bladder.

It may be added here that not all large gall-stones cause death, after their entrance into the bowel, by obstructing it. They some- times become encysted in a pouch which they have themselves been instrumental in producing. Dr. George Harley[1] records a case in which a gall-stone became thus lodged in the duodenum. Some- times, again, they escape *per anum.* It is of course impossible to lay down any law as to the limits of size beyond which it is impossible for a solid body to pass through the ileo-cæcal orifice; but there are good grounds to suspect that in most cases where large calculi have been voided, they have passed by ulceration directly from the gall-badder into the colon.

No distinction need be made between the treatment of cases of obstruction by gall-stones, and that of cases of enteritis.

VI. INTUSSUSCEPTION.

(a) *Pathology.*—By intussusception is meant the prolapse or slipping of a tuck of intestine into the cavity of the portion of intestinal tube immediately below it, wherewith it is continuous. In con- sequence of this, we find the normal course of the intestine in- terrupted by a kind of knot, in which three successive lengths of bowel lie almost concentrically one within the other; the innermost length being formed by the portion of bowel which has descended, the outermost length consisting of the portion of bowel into which the descent has occurred, the middle or intermediate length being the portion of bowel which unites the upper extremity of the one with the lower extremity of the other, and lies therefore in an inverted and everted position between them. The mesentery of the inner two, or included, lengths of bowel is in their descent necessarily dragged down with them into the pouch which they form, and by the unilateral traction which it exerts necessarily gives to their double tube a cur- vature of which the concavity corresponds to the line of mesenteric attachment; so that the lower orifice of the invaginated portion of bowel, instead of lying in the axis of the containing bowel, faces and rests upon some portion of its circumference. The several layers are generally more or less convoluted (with convolutions running transversely) or twisted: but this convolution or twisting is always most marked in the middle tube. The immediate effects of intussusception are, first, more or less obstruction to the passage

[1] Path. Soc. Trans. vol. viii.

of the intestinal contents, and, second, more or less obstruction to the return of blood from the inner two cylinders of bowel involved, to which the stretched and constricted portion of mesentery belongs. It is obvious that the innermost tube must be pretty tightly compressed by the tubes external to it, a condition which must be much increased by the swelling of parts which speedily takes place; especially it is always found to be very tightly girded at its point of entrance by the tumid ring formed at the junction of the outer two layers. Nevertheless, the obstacle which an intussusception opposes is often incomplete, for it is certain that in a good many cases fæcal matters, not always in small quantities, pass through it [pretty constantly; a circumstance due, in part, to the efficiency of the contractile force of the bowel above to squeeze a portion of its contents into the narrowed tube below, but chiefly to the retention still of contractile power in the affected portions of bowel. Very soon after the occurrence of intussusception all the tissues of the inner two tubes, internal to the serous membrane, become black or nearly so with congestion and escape of blood into their substance, and the serous surface consequently assumes a more or less deep slate-colour. At the same time, partly from the accumulation of blood, partly from the transudation of serum, their walls become very greatly swollen, and sanguinolent serum or blood becomes effused from the mucous membrane, and may be found collected both in the interval between the opposed mucous surfaces of the outer two layers of the intussusception, in the central canal, and in the bowel below the seat of disease. At a somewhat later period coagulable lymph is secreted from the opposed serous surfaces of the middle and internal layers, and these become consequently agglutinated in their whole length. The two invaginated tubes remain sometimes for a long while in the condition above described, but often ere long become gangrenous, and then, if the patient survive sufficiently long, separate from their attachments and become discharged *per anum.*

Intussusception is doubtless always an accident of sudden occurrence in connexion with some violent spasmodic action of the portion of bowel which becomes prolapsed. It seems certain, however, that there must be some associated conditions which concur with spasmodic action in producing it. A wave of peristalsis is made up of two distinct elements: first, the contraction of the longitudinal fibres which shortens the bowel and dilates it, and (since it travels from above downwards) draws the portion of bowel below, in which the contraction is commencing, towards the portion of bowel above, in which the contraction is completed; second, immediately following upon this, the contraction of the circular fibres which narrows the bowel and elongates it, and, in elongating it, projects the narrowing segment forward. Now, it is obvious that in these two associated elements, namely, the dilatation of one segment of the bowel with a tendency in its lower part to be drawn upwards, and the narrowing of the segment of bowel immediately above

with a tendency in its lower end to be pushed forwards, we have conditions which, with very slight modification or exaggeration, might permit of the protrusion of the narrowing segment above into the dilated segment below. The circumstances which either one or in combination might have this effect would seem to be: first, the presence of much gaseous matter leading momentarily to excessive distension of the portion of bowel into which the wave of circular contraction is advancing; second, immobility from whatever cause of this distended portion of bowel, so that it is not pushed on bodily by the elongation of the narrowing segment above; and third, the occurrence at this moment of some violent muscular effort, involving the action of the muscular parietes of the abdomen. The sufficiency of these, or of equivalent circumstances, in causing descent of the bowel, is shown in the cases of prolapse of the rectum, and prolapse of bowel through an artificial anus; as well as in the most common case of intussusception, namely, that in which the extremity of the ileum slips into the cavity of the cæcum. It is supposed that the presence of lumbrici occasionally determines the occurrence of intussusception, and with more reason that the presence of a large polypus has this effect. It may be remarked, however, that in some of the recorded examples of concurrence of intussusception and polypus, the intussusception and polypus have been at a distance from one another.

In every case an intussusception must obviously in the first instance involve a short length of bowel only; but for the most part it rapidly increases in size owing to the active peristaltic movements of the several segments engaged. This increase takes place partly by the prolapse of more and more bowel from above, but chiefly by the involution of more and more of the outer layer. In most cases indeed, if not in all, the parts which in the first instance formed the margins of the lower orifice of the invaginated portion of bowel continue to form that orifice, no matter what length the intussusception may ultimately attain. The growth of the innermost tube therefore is the result simply of the descent of more and more bowel from above, while the growth of the middle tube takes place at the expense of the outermost tube only, in consequence of its gradual inversion.

The length of bowel involved in an intussusception varies within wide limits. Including in our measurement the inner two layers only, or those which constitute the intussuscepted portion, the length varies from two to three inches up to three or four feet. A case indeed is quoted by Dr. Peacock,[1] in which, judging from the combined lengths of portions which escaped from time to time *per anum*, there is reason to believe the invagination had comprised twelve feet of bowel.

Intussusception is rather more than twice as common in males as in females, both before and after puberty. It occurs at all periods of

[1] Path. Trans. vol. xv.

life, but is singular, amongst obstructive diseases, in the freque
with which it affects young children.

Intussusception is not very infrequently met with after death
persons (children and adults) in whom during life there had been
reason to suspect its presence, who have had no symptoms th
can be attributed to it, and who have died of some totally d
ferent disease. In these cases the intussusceptions are always fo
in the small intestine—sometimes, indeed, two or three are met w
in the same case—they are generally not above an inch or two l
are easy of reduction, and present little or no œdema or congesti
It is not impossible, as has often been suggested, that similar sl
intussusceptions take place occasionally during good health,
having caused symptoms of more or less severity, undergo sp
taneous evolution with restoration of the integrity of the b
Intussusceptions which prove fatal may occur in almost any par
the intestinal canal, but they occur in different regions, with
different degrees of frequency. Out of 100 fatal cases (accord
to Dr. Brinton's figures), 4 are jejunal, 28 iliac, 56 ileo-cæcal (th
is, involving the cæcum together with the ileum and colon),
12 colic, or originating in and involving the colon only. It m
be noticed, however, that recoveries with separation of the int
suscepted bowel are much more numerous in those cases in wh
intussusception occurs in the small intestine than in those c
in which it involves the colon, a fact which renders it more th
probable that the jejunal and iliac varieties form a larger proport
of the whole number of cases of intussusception than Dr. Brinto
figures might lead us to believe. It may be added, moreover, th
intussusception occasionally begins in the rectum, of which Dr.
Fagge quotes an example; and that prolapsus of the rectum, whi
in some cases involves the descent of the muscular wall together w
that of the mucous membrane, is under these latter circumstance
true intussusception.

Jejunal or iliac intussusception is met with generally, if not exc
sively, in adults. The average age of its occurrence is, according to
Brinton, 34·6 years. It is here that the peculiar curvature of the
vaginated part, due to the traction of the mesentery, is most obs
vable; and it is here, owing probably to the comparative narrown
of the tube into which the invaginated portion of bowel descer
that strangulation and congestion are most speedy and most inte
and that sloughing and separation of the strangulated part are c
sequently most frequent. The length of bowel engaged in th
form of invagination, although it may be as much as several feet
generally less than in intussusceptions involving the large intestine

Ileo-cæcal invagination occurs largely amongst young childr
including babes of a few months old. Dr. Brinton considers that h
the total number of cases are in children under seven years of a
and that the mean age of those affected by it is 18·57 years
begins with the descent into the cavity of the cæcum of the lips

cæcal orifice, which form henceforth the lower extremity of
ʒination. As this increases, the descending ileo-cæcal orifice
ʋn with it more and more of the ileum to form the central
d inverts first the cæcum, and then a gradually increasing
of the colon, to form the inverted or middle layer; and still
ng, finally in some cases reaches the rectum or even protrudes
ᵼ anus. It may be added that the orifice of the vermiform
necessarily retains its position relatively to the ileo-cæcal
nd that the process itself therefore lies at the bottom of the
ʋtween the inner and middle tubes. In ileo-cæcal invagina-
ich is that in which the greatest length of bowel may be
there is generally much transverse folding of the several
intestine which form it, especially of the middle layer, which
ften much convoluted or twisted. Strangulation is compara-
uch more rare here than in intussusception limited to the
ʒestine, doubtless because of the comparative roominess of the
nd in a proportionate degree sloughing and discharge of the
ted tissues are necessarily uncommon.

iety of ileo-cæcal invagination of very rare occurrence is that
ᵼ the lower extremity of the small intestine descends into the
hrough the ileo-cæcal orifice; the lips of the orifice not neces-
ʒscending with it. Strangulation in this case is said to be
ɣ sudden and complete, in consequence of the tightness with
ɪe prolapsed bowel is gripped by the valve. Colic and rectal
ʒeptions are comparatively infrequent, and differ little, except
ɪrts involved, from the ileo-cæcal form of the affection.

ᵼ patient survive sufficiently long after the formation of an
ʒeption, events take place in connexion with it which have
been briefly indicated. The peritoneal inflammation which
ʼoducts unites the opposed serous surfaces of the inner two
nay spread beyond its primary seat, and cause more or less
peritonitis. Or, after these two layers have become united,
ɾ descent of bowel may take place, producing what is called a
ntussusception—an intussusception, that is to say, in which
ʒl above has slipped in the form of a second invagination into
l of the primary invagination. Or, again, as Dr. Aitken[1] shows,
emity of the curved invaginated portion of bowel may, by the
pressure which it exerts against the side of the containing
use at the seat of pressure ulceration and perforation of the
ɪl wall. But by far the most interesting and important event
ɔughing and separation of the included layers of bowel. It
ɪ shown that almost immediately after the occurrence of
tion, these become œdematous, intensely congested, and infil-
ith blood; and it might be supposed from the obstruction to
ɪe vessels supplying them are exposed, that their death must
ily speedily ensue. In many cases, however, patients live for
nd even months, after the occurrence of invagination, with no

[1] The Science and Practice of Medicine, vol. ii.

heless certain characteristic symptoms, which if present point pretty certainly to the existence of the lesion in question.

The commencement of intussusception is attended with sudden and more or less severe abdominal pain of a griping or twisting character, which is referred usually to the neighbourhood of the umbilicus. This generally ceases after a short time, perhaps a few hours, and then after an interval of comparative or total ease returns temporarily, and thus perhaps continues to recur remittently. There is not necessarily any abdominal tenderness, and indeed the patient frequently finds relief, as in colic, by various contortions of the body and by pressure upon the abdominal parietes. Sympathetic vomiting may be an early symptom, but is often in the beginning absent. Constipation generally follows upon the sudden attack of pain : not however immediately, for the bowel below the seat of lesion may, and does generally, continue to act upon its contents until they are completely expelled : nor necessarily, because, as has been pointed out, the intussusception does not in all cases entirely prevent the passage of fæcal matters from above; and sometimes, indeed, instead of any tendency to constipation there is actual diarrhœa. There is one peculiarity, however, in connexion with the intestinal evacuations, which is rarely absent ; it is, that very soon after the occurrence of intussusception, the blood which escapes from the deeply congested mucous surface of the invaginated bowel mingles with the contents of the bowel below, and escapes with them by stool in greater or less abundance.

The symptoms which mark the subsequent progress of the case depend partly on the situation of the intussusception, partly on the degree in which the bowel is strangulated. It has been shown that when the intussusception involves the large intestine, actual strangulation occurs somewhat rarely, and the case tends to become much protracted. In this event the symptoms are apt to be very ill-defined : the paroxysms of pain are often slight, and recur at distant intervals ; constipation may exist at the beginning only or may occur from time to time, or it may never be distinctly present ; there is generally more or less vomiting. As the case, however, progresses, the pain often increases in severity ; the vomiting becomes more and more incessant and possibly stercoraceous; the alvine evacuations either continue to pass or become re-established, blood and mucus are discharged in variable quantities, and even dysenteric diarrhœa comes on. And then after a longer or shorter period, sometimes after two, three, or four months, the patient, who has been gradually getting more emaciated and feeble, dies of simple exhaustion. When the invagination occupies the small intestine, strangulation is usually of rapid occurrence, and its occurrence adds to the symptoms of mere intussusception those of enteritis. The case, therefore, speedily assumes a very threatening aspect. Febrile symptoms manifest themselves, the abdomen becomes tender, incessant vomiting comes on, and the bowel becomes obstructed, or at all events discharges only those matters which the congested and gangrenous tissues pour

out. Under such symptoms, the patient, as in uncomplicated enteritis or internal strangulation, may speedily succumb; but sometimes, at a moment when the disease appears to be still progressing unfavourably, the constipated bowel begins to act, offensive stools mixed with blood and mucus begin to be discharged with more or less tenesmus, vomiting diminishes or ceases, febrile symptoms abate, and after a longer or shorter period of dysenteric symptoms a sequestrum is passed *per anum* in the form of a dark fœtid gangrenous mass.

The most characteristic features, amongst those which have been enumerated in the symptomatology of intussusception, are, first, the sudden onset of the malady, with pain and more or less constipation and vomiting; and secondly, the discharge of blood *per anum* which is generally present even from the beginning. But there is a third sign, to which no allusion has yet been made, which is perhaps of even greater importance, namely, the presence of a tumour. It can scarcely happen that any length of a threefold tube of intestine, especially when its layers, one or all, are congested and swollen, can be present without forming a tumour capable of detection by careful palpation through the abdominal walls, provided at least these be not too fat or too rigid, or the bowels generally be not too much distended with gas, or the abdominal tenderness be not too great, to admit of satisfactory examination. The presence of a tumour indeed, especially in the case of ileo-cæcal, or colic, invagination, may often be recognised during life; and that the tumour is an intussusception, may also often be recognised, partly by its cylindrical form, partly by its position, but especially by the fact that it may in some cases be detected changing somewhat from day to day in form and direction, as the intussusception increases, and may sometimes also be felt to dilate and harden, and then subside, under the influence of its peristaltic movements. Further, in those cases in which the intussusception extends low into the rectum, its lower extremity may be detected with all its characteristic features by the finger inserted into the anus.

It must not be supposed, from the foregoing observations, that there is always a wide distinction between the symptoms of invagination of the small intestine and those of invagination of the large intestine. There is no doubt that the majority of jejunal and iliac invaginations are marked by the violent symptoms and rapid progress which have been assigned to them, and that the majority of invaginations involving the large intestine present less urgent symptoms and assume a chronic character. But undoubtedly in some cases invaginations of the small intestine approximate in symptoms and in progress to those of the large intestine, and in a still larger proportion of cases cæcal and colic intussusceptions are attended from an early period with symptoms of great urgency and prove rapidly fatal. These differences depend apparently on the presence or absence of strangulation, which, as has been shown, may occur in connexion with any form of invagination, but which generally occurs early

hen the small intestine alone is affected, late and perhaps not at all
hen the large intestine is the seat of disease. And it is important
 bear in mind that it is this very strangulation, leading to engorgement,
flammation, and gangrene of the invaginated tract of bowel, which,
hile it gives rise to the most urgent and distressing symptoms and
ot unfrequently induces speedy death, is effecting the separation of
e obstructing mass, and thus leading to the only possible solution of
e case compatible with restoration to health.

There are several additional points in which as a rule differences
vailable for diagnosis are manifested between invaginations of the
nall and large intestines respectively. Dr. Brinton has especially
welt upon them. First, tenesmus is common in invagination of the
rge intestine, but is not necessarily present, and is generally absent
hen the small intestine is affected; secondly, hæmorrhage from the
owel (connected doubtless with the relative degrees of congestion of
e invaginated portion of bowel) is much more copious in invagina-
on of the small intestine than in that of the large, and blood may
lso in the former case be vomited; thirdly, obstruction of the bowels
 a more prominent symptom when the small intestine is affected,
an when the large intestine is affected. The remaining points on
hich Dr. Brinton insists, namely, the situation of the tumour within
he abdomen, and the discovery of the end of the intussusception in
he rectum, have been already discussed.

Hitherto it has been supposed that the case of intussusception has
een uncomplicated with any other malady; but it must not be for-
gotten that general peritonitis may come on at any time in its pro-
gress, and that it is sometimes induced by perforation of the bowel.
The latter event is especially apt to occur at the time of separation
of the slough, and necessarily renders a case, already sufficiently pre-
carious, hopeless.

The percentage of deaths in intussusception must be very large;
it is very difficult, however, if not impossible, to estimate what that
percentage is. The stage at which patients die, and the immediate
cause of death, present very great varieties. Dr. Brinton estimates
that the average duration of cases directly fatal is five and a half
days. This estimate may probably be accepted with regard to those
cases in which strangulation marks the onset of the intussusception,
and generally therefore with regard to invagination of the small
intestine; but, as Dr. Fagge points out, it can only be true, in a
qualified sense, of invagination of the large intestine,—namely, if we
reckon the duration of the case from the first manifestation of
symptoms of strangulation, and not from the moment at which
invagination commenced, which may have been many weeks pre-
viously. In cases in which there is not immediate strangulation,
the patient may survive for weeks or months, ultimately dying of
exhaustion, or killed by the supervention of strangulation. Even
after the slough has been discharged, and the continuity of the
segment of bowel above and that below the neck of the invagination

has been established, permanent recovery would seem to be less frequent than ultimate death,—death being induced at various intervals afterwards, either by exhaustion or by the effects of stricture of the bowel. Recovery after the separation of a portion of the small intestine seems to be more frequent, both relatively and actually, than after the separation of a portion of the large intestine.

(*c*) *The Treatment* of intussusception, like the treatment of other forms of intestinal obstruction, must be on the whole negative; or, to be more explicit, the less actively the patient is treated, the more likely is he to have his life prolonged, and ultimately to recover. Here, as in most other kinds of obstructive disease, all forms of purgatives must be eschewed, everything in fact must be avoided which can have the effect of promoting peristalsis; for violent movements of the bowel, independently of any other mischief they may effect, naturally tend to increase the size of the intussusception. Neither must it be forgotten that the special ground on which alone the administration of purgatives may be urged exists less in intussusception than in other forms of obstructive disease; for constipation is rarely complete at any rate for more than a few days. On the other hand, opium is of extreme value for the sake both of relieving the pain due to enteritis, or to violent peristalsis, or both, and of restraining the exaggerated movements of the bowel. Dr. Brinton suggests that belladonna, on account of its relaxing influence on the unstriped muscular fibres, may be given with advantage, either alone or combined with opium. Enemata are often beneficial, partly by relieving the lower bowel, partly, perhaps, by acting as a kind of internal fomentation. They may, however, possibly have another value, at all events when administered in large quantities, gradually and without violence. Thus there is some reason to believe that where the large intestine is affected, the distension caused in the external tube of the intussusception, and the pressure exerted on the invaginated portion of bowel itself by such injections cautiously administered, may in some cases, especially those of recent origin, and where the length of bowel involved is as yet small, avail to effect its restoration. Inflation of the bowel *per anum* was long ago recommended for the same purpose; and of late years this procedure has been revived, and several cases have been recorded in which it seems to have been successful. It is obvious, however, that, as is the case with ordinary enemata, inflation can only be of service when the invagination involves the large intestine, and when it is in an early stage. But in intussusception, as in other forms of disease attended with obstruction, the question of surgical interference is not unlikely to arise—Can any surgical operation be performed with a prospect of benefit? It may be supposed that it would be no difficult matter, after opening the abdominal cavity, to withdraw from its sheath an intussuscepted portion of bowel; and no doubt, if adhesions had not yet been formed, or if gangrene had not yet taken place, the evolution of the intussusception might be effected; yet even then considerable

force would have to be applied, especially if the intussusception were large, and much risk of damage would attend the process of retraction. Assuming then that an operation might under certain conditions be attended with advantageous results, the question as to what these conditions are naturally presents itself. Now, considering how acute are the symptoms which attend invagination of the small intestine; how speedily adhesions, gangrene, and separation of the slough begin to take place in it; how difficult it is to feel sure of the nature of the case at that early period when alone an operation would have a chance of success; and moreover how often (comparatively) the patient is restored to health by the spontaneous discharge of the invaginated length of bowel,—it seems scarcely possible to avoid the conclusion that in these cases at least surgical interference should be discarded. But when, on the other hand, we bear in mind that, in intussusception of the large intestine, ultimate recovery is exceptionally rare, even after the separation of the invaginated portion of bowel, that this separation is of very infrequent occurrence, and that the invaginated bowel is apt to remain in a fairly healthy condition for weeks, sometimes, after the commencement of the disease, it is obvious that we have here an opportunity for operation and a chance of benefit from it very much more favourable than those which iliac and jejunal intussusceptions offer. And it becomes difficult not to accept the conclusion to which Dr. H. Fagge comes, which is to the effect that it is in these cases, and in these alone, that the question of operating should be seriously entertained.

Concluding Remarks.

Before finally dismissing the subject of intestinal obstructions, it may be convenient to consider, however briefly, some of the more important points upon which our discrimination of such cases of obstruction as may come before us must mainly depend, as well as some of those points of treatment which have a general value in reference to them.

(a) *Pain* is a more or less general and prominent symptom in all cases of obstruction, but it varies a good deal in different persons, both in duration, character, and severity. It is partly the pain of peritonitis, partly that of colic, and these may be present separately, or variously combined. Hence it can be readily understood, that although in well-marked cases the character of the pain may afford us valuable assistance in determining whether the peritoneal surface is alone diseased, or whether the inflammation affects the inner tunics only of the bowels, or whether it involves pretty equally the peritoneal, muscular, and serous coats; in others it affords no evidence whatever of a trustworthy kind. I have a distinct recollection of one of the most extensive and severe cases of enteritis I ever saw, associated with peritoneal inflammation, which a quite well-experienced

medical man regarded almost to the last as one of simple colic. It may be added, that even where there is distinct inflammation of a length of bowel, the pain and tenderness, instead of occurring immediately superficial to the affected gut, are frequently most marked in the umbilical region. This latter peculiarity is manifested not unfrequently in cases of inguinal or femoral hernia, and is, indeed, a not uncommon characteristic of affections of the small intestines.

Painful peristaltic movements coming on in paroxysms constitute one of the most distressing, and at the same time one of the most characteristic, symptoms attendant on obstruction; yet, although they may be present in a marked degree in all forms of obstruction, I agree very much with Dr. Fagge in the belief that they are for the most part most severe and most constant in the cases of longest duration; in the cases, therefore, in which enteritis is either not present at all or occurs late.

(*b*) *Vomiting* is rarely if ever absent from the various affections now under consideration. In the beginning it is sympathetic only, and in that respect resembles the vomiting which attends many other affections not necessarily involving the gastro-intestinal tract. After a while, however, in most if not all cases, it owns a more direct cause. The bowels above the seat of obstruction become distended with contents, partly with what has been taken by the mouth and has been transmitted onwards, partly, as Dr. Brinton justly insists, with the secretions of the intestinal walls; these, by the combined effects of simple overflow, of the peristaltic movements of the bowels and of the pressure exerted on the bowels from without, gain an entrance into the stomach, and then become vomited, constituting what is called stercoraceous vomit. The stercoraceous matter, though never in cases of simple obstruction derived from the large intestine, and probably never directly from the lower part of the small intestine, still acquires a thin pea-soup-like aspect and a fæcal odour, which the normal contents of the stomach never do assume, and which are doubtless the result simply of the long residence of the intestinal contents within the bowels, and of their admixture there with bile and other secretions. Vomiting is generally an early symptom in all cases of intestinal obstruction, and in those of acute progress may continue to the end without intermission. Yet even in some of these it intermits, and may be absent for a comparatively long period. In the more chronic affections its occurrence is extremely variable; but even here vomiting generally becomes more or less constant, and then stercoraceous towards the close of life. There is no doubt that vomiting is an earlier, a less interrupted, and a more severe symptom, in proportion to the nearness of the seat of obstruction to the stomach, and that for this reason it is a more marked accompaniment of obstruction of the small intestine than of obstruction of the large.

(*c*) *Constipation* is naturally one of the most characteristic phenomena of obstructive disease, and its occurrence is of high diagnostic value; yet it need scarcely be repeated that fæcal matters will often

ass with comparatively little difficulty through even a tight stric-
ture, especially one in the small intestine; nor must it be forgotten,
that generally at the time at which complete obstruction is established,
the bowel below contains a larger or smaller quantity of fæces, which
may be removed naturally or by injection, and the removal of which
might lead to the belief that no obstruction exists. Scybala may
sometimes be seen in the large intestine, after death from complete
obstruction of the ileum of many weeks' standing. Nevertheless,
constipation of an insuperable character is for the most part an
exceedingly pronounced symptom; coming on suddenly, and persist-
ing in cases of internal strangulation, and of the lodgment of gall-
stones; coming on somewhat gradually, or at all events with
premonitory stages, in most cases of stricture and of compression. In
intussusception there is also generally sudden constipation, of various
duration, but the invaginated mass (especially when the large intestine
is involved) is rarely quite impervious, so that before long a slight
transmission of fæcal matter begins, at all events in all chronic cases,
to take place; moreover, in cases of intussusception, blood is usually
passed by stool at an early period, and more or less continuously
throughout their whole duration. The discharge, indeed, from the
large intestine assumes something of a dysenteric character, and
becomes associated with symptoms in some respects resembling those
of dysentery. In intussusception of the small intestine, the dis-
charge of blood is sometimes very copious.

(*d*) *Tumour and Shape of Belly.*—The belly in cases of obstruction
soon becomes more or less tense and tympanitic (unless, indeed, the
obstruction be in the upper part of the small intestine) in consequence
of the distension of the bowel above the seat of stricture by accumulated
fæcal matters and by gas; and in some instances the shape which the
abdomen then assumes may aid in the diagnosis of the site of obstruc-
tion. Thus, if the rectum were blocked up, distension, though soon
extending throughout the whole of the large intestine, would first take
place and be most extreme in the sigmoid flexure and descending
colon, in the situation of which parts, therefore, some special fulness
might be looked for; if the obstruction existed in the transverse colon,
some fulness would not improbably be discovered in the right flank,
and, according to circumstances, in the position of a larger or smaller
portion of the transverse colon, the left flank presenting a comparative
absence of fulness, tension, and even perhaps of weight; while, again,
if the impediment occupied the lower part of the ileum, the distension
would probably be most marked in the mid region of the abdomen.
But, as has been before pointed out, the evidence afforded by the general
shape, and resistance, and weight of the abdomen must be received
with great caution, for the distended bowels very readily deviate from
their usual position, and diffuse themselves, as it were, beneath the
abdominal surface, displacing, or at least concealing the bowels, which
are collapsed and empty. Sometimes, indeed, in distension of the
large intestine, the sigmoid flexure extends over the whole front of

the abdomen, and with the aid of the other lengths of colon effectually conceals the whole of the small intestine from observation. The presence of an abdominal tumour, as distinguished from mere distension of bowel, is an important element in diagnosis. It need scarcely be said that, in internal strangulation, and in most cases of compression, no tumour is likely to be felt; and indeed in stricture also, unless the stricture depend on some form of cancerous growth, or be associated with the presence of peritoneal cancer, or be in the rectum within reach of the finger, no tumour will probably be distinguished. In cases of lodgment of gall-stones, the lump produced by the presence of the gall-stone might, one should suppose, be not very difficult of detection; but unquestionably in the great majority of them, of those even under the care of thoroughly competent practitioners, no tumour has been recognised during life. Indeed it may be pretty confidently asserted that they are rarely, if ever, recognised. This fact may be due in some degree to the absence generally of very minute manual examination; but it must not be forgotten that the tumour formed by a gall-stone is really not very large, that the swelling of the bowel above the obstruction tends to cause the point of obstruction to recede from the surface, or to mask it, and that tenderness, abdominal fatness, rigidity of muscles, and other conditions, all aid more or less to interfere with successful manual examination. Of all the different forms of obstruction which have been enumerated, intussusception is the one which is most commonly attended with the presence of manifest tumour; but tumour seems to be far more common in connexion with intussusceptions involving the large intestine than in that form of the disease which is limited to the ileum and jejunum. It is needless to repeat the characteristic features which such tumours present.

(e) *The Condition of the Urine* has been regarded, ever since Dr. Barlow's[1] interesting observations on the subject were published, as some indication either of the seat of obstruction, or of some other conditions connected with the obstruction. Dr. Barlow observed that, in a case of his, in which the obstruction was in the duodenum, there was an almost total suppression of urine; and there is no doubt that in many cases of obstruction high up, the same phenomenon is manifested. He argued that the great diminution of this secretion, in his and in similar cases, was caused by the constant vomiting which is always present in obstruction of the upper part of the small intestine, and by the little available absorptive surface which is presented, combining to prevent the entrance of fluid into the vascular system, and the supply of an adequate amount to the kidneys for the maintenance of their secretion. And he argued further, that the abundant discharge of limpid urine which is frequently observed in cases where the seat of obstruction is low down, is to be explained by the presence of entirely opposite conditions. Further observation, however, seems to show that although there may be a tendency on the whole to a dimi-

[1] Guy's Hospital Reports, vol. ii. Second Series.

hed secretion of urine when the impediment is high up, and to an
reased, or at all events fairly abundant secretion when the impedi-
nt is low down, the urine is in many cases abundant or scanty ap-
rently quite independently of the seat of obstruction. Dr. Brinton,
eed, suggests that the diminished secretion of urine which is
quently met with, and the variability of which phenomenon he
ly recognises, is rather due to a kind of vicarious secretion into
e bowel above the seat of obstruction, to which also, rather than to
resta, he no doubt rightly attributes most of the distension of the
wel and much of the vomit. Mr. W. Sedgwick,[1] however, apparently
th more reason, argues that the diminution or suppression of the
inary secretion is related to the suddenness and intensity of the symp-
ns, and is immediately due to the reflected influence of the abdo-
inal sympathetic centres. On the whole, even if we adopt Mr.
dgwick's views, it may probably be accepted as generally true that
minished secretion of urine—often, however, temporary—attends
ose cases in which the symptoms are of sudden occurrence and
ute; and that a fairly abundant secretion of this fluid characterises
ses which are chronic in their course; and that, mainly on these
ry grounds, suppression or diminution of urine is far more com-
on in cases in which the small intestine is obstructed, than in
ose in which the impediment occupies the larger bowel.

(*f*) *The Mode of Invasion* is often of great value in reference
diagnosis. Internal strangulation and intussusception always
gin suddenly, with more or less acute and severe symptoms.
bstruction by gall-stones might be expected to be preceded by
mptoms indicative of the passage of a gall-stone from the bladder
to the duodenum, and by further symptoms arising in the course of
s journey to the spot at which it becomes finally arrested; and some-
mes, but by no means always, the history of such premonitory
mptoms can be pretty clearly obtained. Stricture, on the other
nd, and in a less marked degree obstruction from compression of the
wel, are in the great majority of cases preceded for a more or less
nsiderable length of time by symptoms which point to what is
ing on, and which for the most part have a resemblance to those
hich attend the fatal attack.

(*g*) *The Duration of Life* after the commencement of symptoms
hich lead to the belief in the presence of one of the maladies under
nsideration varies considerably in different cases. The continuance
life is compatible with the persistence of mere, though complete, colic
rectal obstruction of several weeks' or even months' duration. But
ath as a rule supervenes much earlier in proportion as the impedi-
nt is situated nearer to the stomach. When, however, enteritis is
sociated with obstruction, then, wherever the obstruction may be, the
ogress of the case is always very rapid, and, dating from the com-
encement of enteritic symptoms, rarely occupies more than a week,
en only three or four days. Hence internal strangulations, obstruc-

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[1] Med.-Chir. Trans. vol. li.

tions by gall-stones, and intussusceptions in which strangulation occurs (more particularly therefore intussusceptions of the small intestine), are usually fatal within a few days after the commencement of symptoms; while obstructions from stricture or compression, and generally also those from intussusception affecting the larger bowel, for the most part present a comparatively chronic progress.

(h) *Statistics.*—There are certain striking facts deducible from the statistics of obstructive diseases, which it is always well to bear in mind. First, as regards age and sex. It is a well-ascertained fact that obstruction by gall-stones always occurs late in life, generally over fifty, and about four times as frequently in women as in men; it appears also that intussusception may occur at all ages, and is at all ages somewhere about twice as common in males as in females, but that of intussusceptions involving the large intestine (which form pretty nearly two-thirds of the total number of fatal intussusceptions), probably fully one-half occur in children under seven years of age; it appears further that stricture (if we omit strictures due to congenital malformation) is a disease of adult life and occurs indifferently in both sexes. Next, in reference to the portion of intestine involved. Stricture, as a cause of death, belongs almost without exception to the large intestine, and not only so, but at least three-fourths of the total number of strictures are situated below the middle of the transverse colon; compression and traction belong essentially to the small intestine, and may be regarded, as Dr. Fagge observes, in a practical point of view as the strictures of that tract; internal strangulation occurs more particularly in connexion with the small intestine, or with the cæcum and sigmoid flexure; gall-stones, with hardly an exception, become arrested somewhere in the jejunum or ileum; and the large intestine is involved in intussusception at least twice as often as the small intestine alone. Lastly, with respect to the relative frequency of the several lesions, it may be well to quote Dr. Brinton's figures, based on 500 deaths from obstruction; according to which it appears that out of 100 cases, 43 are cases of intussusception, 17 are cases of stricture, 4·8 are cases of impaction of gall-stones, 27·2 are cases of internal strangulation (including, however, all those cases which have been here described as compressions), and 8 are cases of torsion, in regard to which the opinion has been previously expressed that they are simply cases of uncomplicated enteritis.

(i) *Finally, in respect of Treatment,* there are a few established principles which must guide us in all cases of sudden obstruction of the bowels, and especially in all cases where that sudden obstruction is attended with symptoms of enteritis. First, purgatives however mild can do no good, may do immense harm, and must be altogether discarded. Secondly, opiates and other sedatives must be administered largely or at least sufficiently largely to produce some visible effect in relieving pain and giving rest, and should in most cases be administered by subcutaneous injection. Thirdly, but little food and stimulus should be

istered by the mouth, for they are almost always immediately
:d, or if retained fail to be absorbed, and then add only to the
f fæcal matters distending the bowel above the seat of obstruc-
n either case adding to the patient's distress and tending to
ı death. Food given by the mouth should be in small quan-
fluid, and easy of absorption and digestion. There is no reason,
er, in many cases, why we should not endeavour to support
tient's strength by nutritious enemata. Fourthly, operations for
lief of intestinal obstructions are rarely followed by satisfactory
ı; nevertheless, if there seem a chance, however remote, of
ening the life of a patient who is otherwise doomed to speedy
few would hesitate to catch at that chance. In some forms of
ction an operation must from the very nature of things be at
ıseless, as for example in simple enteritis, in torsion, in most
of compression of the bowel, and in the impaction of gall-stones ;
ere can be no doubt that if an operation were performed at an
date, internal strangulations might be relieved with fair success,
ıtussusceptions might be retracted with frequent benefit. Dr.
is doubtless judicious in recommending an operation for the
tion of ileo-cæcal intussusception, for reasons which have been
previously ; and there can be no doubt that if the evidence
at all strongly to internal strangulation, an early resort to
y should be had. It need scarcely be insisted on that no patient
ng from sudden obstruction with enteritic symptoms, in whom
ernal hernia, whether strangulated or not, exists or has existed,
ı be allowed to die without undergoing an exploratory operation
seat of hernia.

ULCERATION OF THE BOWELS.

By John Syer Bristowe, M.D. F.R.C.P.

ULCERATION of the bowels, using the word in its widest sense to indicate all those cases in which the mucous membrane is partially—no matter how or why—destroyed, is a lesion of very common occurrence, sometimes induced by the extension of disease from the exterior of the intestine, more commonly the result of morbid processes commencing in its mucous and submucous tissues.

I. PATHOLOGY.—(*a*) *Ulceration beginning from within.*—Ulceration which originates in connexion with the mucous membrane may be found at any part of the intestinal tract; but there are certain situations in which it is met with much more frequently than elsewhere: these are the duodenum, the ileum (especially towards its outlet), the cæcum, ascending colon, sigmoid flexure and rectum; in other words, the commencement and the termination of both the larger and the smaller bowel.

The causes of ulceration are very various, and are not always easy to define, and still less easy in practice to recognise. Some forms of it are no doubt distinctly the result of the liquefaction or destruction of some specific deposit, as in enteric fever and in tuberculosis, and, perhaps, in the latter stages of syphilis; and some, as possibly the dysenteric, are due to some specific kind of inflammation. But in a considerable number of cases the causes of ulceration are local; the bowel is wounded by some sharp body which has been swallowed, or is rubbed and irritated by some partially arrested solid mass, or is fretted by the constant passage over it of acrid fluids, or presents some localised point or points of inflammation, which own no more manifest cause than does a pustule of impetigo, a bleb of pemphigus, or an ordinary boil. It may, however, be conceded, that even in these latter cases the general condition of the patient has often much to do, at all events indirectly, with the production of ulceration: that, for example, on the one hand the fluids which irritate are often irritating in consequence of being unhealthy; and, on the other hand, the fretted bowel often inflames or ulcerates under their influence, because it was previously congested, or its circulation was sluggish.

Many forms of inflammation of the skin are attended with an excessive production of epidermis, or with the exudation of matter

beneath the epidermis, and thus become characterised by the
pment of squamæ or of crusts, on the removal of which a more
raw surface is left, and beneath which ulceration is apt to take
 The varieties of cutaneous inflammation, here very briefly
ed, are for the most part easy of separate recognition, yet they
requently merge one into the other. But on mucous surfaces
tinctions between scaly, vesicular, and even pustular affections
rely, if ever, very obvious, the delicacy and moisture of the
lium interfering alike with the formation of a mere dry scale,
ith the limited accumulation of fluid beneath it. I have used
rm "croupous" on another page, to indicate those forms of
nal inflammation in which the mucous membrane is found
d with an opaque adherent film, composed of corpuscular ele-
 derived partly from its surface, partly from its glandular
tions; but I have used it in no specific sense, and believe
in many cases at least, the film, or false membrane, is
ogous with the scurf of pityriasis, the scales of lepra, or the
s of eczema. Ulceration of the bowels not infrequently com-
s with "croupous" inflammation: a linear or irregularly polygo-
stellate patch of more or less intense congestion and tumefaction
 its appearance, which soon becomes covered (excepting, perhaps,
edges by which it may be extending) with an opaque whitish
f-coloured exudation, which is somewhat friable and granular on
irface, and extends by rootlets into the Lieberkühnian follicles;
tch of exudation after a time separates, and leaves sometimes a
 surface, sometimes a slight excoriation, or even a distinct ulcer,
ested by a somewhat cupped greyish or yellowish surface and a
narked margin of congested mucous membrane. Ulcers com-
ing thus may be met with in any part of the bowels, but are
 more common in the large intestine than elsewhere. In the
intestine they chiefly affect the free edges of the valvulæ con-
tes, and in the large intestine either the projecting ridges formed
e intervals between the sacculi, or those which correspond to the
udinal muscular bands. They are very apt to occur, particu-
in the large intestine, in the course of pneumonia, and in cases
iich the patient is dying from many forms of chronic disease,
 as Bright's disease of the kidneys, cirrhosis, cancer, chronic
sis; and, from the peculiar position which they occupy, there is
n to believe that they depend, partly at least, on irritation by
utestinal contents. Occasionally we find large tracts of bowel
or less deeply congested, and studded with irregular patches or
s, or an imperfect network, consisting partly of croupous exu-
n, partly of consecutive ulceration.
 other cases ulceration commences either from distinct me-
cal injury or from more gradual erosion; the ulcer then
 roundish, or more or less irregular in form, varies in size,
nting a more or less congested and well-defined, but not neces-
 thickened, margin, and a more or less irregularly excavated

shreddy greyish surface. Such ulcers may be observed when gall-stones or other solid bodies have lain for some time in contact with a portion of intestinal surface ; they occur also in the large intestine, when it has been long distended with accumulated fæcal contents. In several cases of long-continued constipation, I have seen the mucous surface of the larger bowel studded with tracts varying from about one to twelve square inches in area, consisting of groups of circular ulcers of the kind now under consideration from half an inch downwards in diameter, and separated from one another by a net-work formed of congested and partly undermined bands of mucous membrane.

Sometimes, again, ulcers obviously originate in patches of sub-mucous suppuration, as we see occasionally in pyæmia, or in patches of submucous slough, like an ordinary furuncle. Among these may, perhaps, be reckoned the ulcerative inflammation of the follicles of the colon, which Rokitansky describes, and which seem by many to be considered the earliest stage of dysentery. The follicles first enlarge to between the size of a tare and a pea, and become sur-rounded by a dark red halo of congestion, and then, undergoing sup-puration, discharge their contents into the bowel by an ulcerated opening, which eventually enlarges, and forms a circular ulcer with overlapping edges. When the follicles are widely affected, the mucous membrane presents in the first instance a generally congested tuberculated surface, upon which, after a short time, groups of small tolerably deep circular ulcers make their appearance.

In other cases, again, ulceration is produced by the separation of a slough. In various parts of the small intestine, but perhaps most commonly in the duodenum and jejunum, as well also as in the œso-phagus and stomach, circumscribed patches of intense congestion or of extravasation of blood appear in the substance of the mucous mem-brane, the patches shortly dying, and coming away either bit by bit or in mass. The formation and separation of such patches are often effected with little obvious change in the parts immediately surrounding them ; there is often no unwonted congestion observable, and the pits which are formed by their removal for the most part speedily become effaced. I believe they are most commonly seen in cases of small-pox, typhus, and other such diseases. A somewhat similar condition is sometimes observed in the valvulæ conniventes, and still more frequently in the transverse projections from the interior of the larger intestine, the free edges of which then present a line of ulceration, which looks as though it had been formed by a mere splitting of the diseased mucous membrane, and presents either an ashy or a yellow flocculent surface.

But sloughing to a much more serious extent is sometimes met with, especially in the large intestine ; patches of surface become livid, or brown, or nearly black with congestion, and then their central region assumes a grey or ashy colour, gets shrunken, depressed, and softened, and soon breaks down into a soft shreddy substance, which partly becomes detached and partly adheres to the floor of the

ation, and to the not yet broken down edges, which latter tend to
l, and to involve more and more of the surrounding tissues.
ionally extensive tracts of the mucous surface of the large in-
e are covered with sloughing patches, originating in the manner
escribed.

is not pretended that all non-specific ulcers arise in one or
of the modes here enumerated, or that the several varieties enu-
ed are even in the beginning in all cases essentially distinct
one another. Still less do they necessarily maintain these
ctions in the later stages of their progress. Fully formed ulcers
d present a considerable variety of appearance, dependent mainly
e processes which are taking place in them. Thus, when they
a process of healing, we find the general surface smooth and
, or it may be granulating, the edges little if at all thickened or
sted, perhaps puckered, and sloping more or less obviouly to
urface of the ulcer with which they are continuous; when they
luggish, the edges are more or less tumid and rounded, and it
be overhanging, and the general surface smooth, or somewhat
ilar and flocculent; and again, when they are spreading, the sur-
ling mucous membrane presents more or less intense congestion
welling, and the immediate edge of the ulcer is either flocculent
ish-coloured, or presents a vivid red, raw, bleeding wall, or forms
re or less complete rim of distinct gangrene. The floor of an
inal ulcer is generally constituted by the submucous tissue, but
nfrequently the transverse muscular fibres are distinctly exposed,
ially in an ulcer which is still spreading; and when the ulcer
to perforate the bowel, the muscular coat itself becomes opaque,
d, and in parts destroyed.

e account just given applies to individual ulcers. But very
ently, and much more frequently in the large than in the
l intestine, numerous ulcers are present at the same time, and
to increase either in number or size and to coalesce in a greater
ss degree; and then, according to the stage to which the ulcera-
has advanced, we meet in different cases with either a number
undish ulcers separated by an imperfect network of mucous
brane, or interlacing networks of ulceration and of mucous
brane, or islets of mucous membrane in an expanse of ulcera-
or lastly, extensive tracts from which the mucous coat has
wholly removed. In these cases the transverse muscular fibres
ften freely exposed, and the remains of mucous membrane are
nd swollen and rounded, and form tubercular excrescences. The
l, moreover, is frequently much contracted.

me of the specific forms of intestinal ulceration have been
here considered. There is only one, indeed, tubercular ulcera-
which needs anything like minute description here. Still it
be convenient briefly to advert to some of the more im-
nt features which do, or are supposed to, distinguish them
ally. I am not aware that syphilitic ulceration has been

surely recognised in the alimentary canal, except in the neighbourhood of its inlet and outlet; intestinal ulceration, however, is often met with in persons who have died when under the influence of the syphilitic virus, and it seems at least reasonable to suppose that in some of these cases the ulceration, even though it presents no visible distinctive mark, owns a syphilitic origin. Dysenteric ulceration occupies the large intestine, and occasionally invades also the lower part of the ileum. The mode of origin of the tropical form of the disease is variously described; by many, including the late Dr. Baly, it is considered to arise in inflammation and suppuration of the solitary glands; by others it is believed to originate in a croupous form of inflammation; and no doubt it sometimes commences with intense general inflammation, passing at once into gangrene. But, however it may begin, it tends to the rapid destruction of extensive tracts of mucous membrane, and to that chronic condition of more or less extensive rawness which has been above referred to. In typhoid fever a deposit takes place in the solitary glands, and in Peyer's patches (more frequently in the latter than in the former), which become congested, softened, and form flat wheal-like elevations. At the end of a few days, it may be a week, the bulk of the enlarged gland begins to slough, a line of ulceration forms around the slough, and this latter acquires a peculiar yellow or brownish hue. In a short time the slough separates, leaving a circular or sinuous ulcer with congested tumid edges, and an excavated surface, limited either by the submucous tissue or by the transverse muscular fibres. Then usually the edges begin to resume the normal thickness and colour of mucous membrane, and to blend gradually with the contiguous surface of the ulcer, which itself fills up and contracts, and ultimately heals with a scarcely or not at all visible cicatrix. At other times the ulcer remains irritable or sluggish, or spreads both in surface and depth, either by gradual erosion, or by sloughing, or by the phagedænic process. And then sometimes hæmorrhage, sometimes perforation of the bowel takes place. Typhoid ulcers vary in size from about that of a split pea to that of the largest of Peyer's patches. They are always most marked immediately above the ileocæcal valve (to which part they are sometimes limited), and extend thence, gradually decreasing in number and size, upwards through the ileum and occasionally the jejunum. They occur in the large intestine in about half the total number of cases, being then of smaller size than those in the ileum, and diminishing in number from the cæcum downwards.

Tubercular disease of the mucous membrane of the bowel is one of the most frequent forms in which the tubercular diathesis reveals itself, and certainly the most frequent cause of intestinal ulceration. It occurs in rather more than one half of the total number of cases of pulmonary consumption, and rarely if ever independently of it; and it is often associated with peritoneal and other varieties of abdominal tubercle. It affects primarily the same structures as are affected in

enteric fever, namely Peyer's patches and the solitary glands; and in the small intestine therefore is always most advanced and most abundant immediately above the ileo-cæcal valve, from whence upwards (although it may extend throughout the ileum and jejunum) it gradually diminishes. It affects the cæcum more than any other part of the large intestine, involving also the ileo-cæcal valve and the vermiform appendage; but it may form patches throughout the whole of the colon. The large intestine and small intestine are affected by it with equal frequency, and they are both affected in combination about twice as frequently as they are each affected separately. The tubercular material is deposited, either in the form of grey granules or of yellow cheesy masses, in the substance of the congested and swollen glands, and generally soon undergoes softening, producing a small pretty deep ulcer with thickened elevated overhanging edges. When several of these deposits have softened side by side, as happens in Peyer's patches, the ulcerated area presents in the first instance a kind of honeycombed appearance, the small ulcers being separated by more or less complete bridles of yet undestroyed and thickened mucous membrane, and the general margin, which is also thickened, presents a sinuous or scolloped outline. Tubercular ulcers generally tend to spread by the successive deposition and softening of tubercles at their edges, the tubercles not being then necessarily limited to the glands; and by this process they often extend over a considerable area. In the large intestine the whole mucous membrane of the cæcum is sometimes thus destroyed, and often very extensive tracts of ulceration are found to stud the surface of the colon at more or less distant intervals. In the small intestine tubercular ulceration has a remarkable tendency to spread in the transverse direction, and frequently forms bands from half an inch to an inch or more wide, occupying the whole circumference of the bowel. Many of these are sometimes met with at short distances from one another throughout the greater part of the small intestine. In most cases the ulcers still go on enlarging up to the patient's death, and occasionally they lead to hæmorrhage or to perforation. Sometimes, however, they cicatrize more or less perfectly: some cicatrizing indeed while others are spreading, or new ones are forming. But, owing to the extensive destruction which tubercular ulceration occasions, cicatrization is generally attended with considerable contraction; so that sometimes in the small intestine, in the cæcum, or in the colon, the calibre of the bowel becomes in consequence so much diminished as to produce a real stricture. Sometimes, again, tubercular deposits dry up or become absorbed without ever undergoing actual ulceration; and it is not a rare thing to find, in cases of chronic phthisis, both in the large and small intestines, small, irregular, elevated patches, sometimes associated with ulceration or the remains of ulceration, which present a dark greyish hue and a cicatrix-like appearance, the surface being studded with small granules, the edges being puckered and prolonged by irregular bands into

the membrane around, an appearance having some resemblance to that produced by superficial lupus. The peritoneal surface corresponding to tubercular ulcers of the mucous membrane is generally studded with minute grey granulations and the lymphatics ramifying in the walls of the same part, and those extending between it and the nearest mesenteric glands are often filled with opaque white creamy or cheesy contents. It may be added that extensive chronic ulceration of the large intestine, which has all the characters previously described as belonging to the later stages of dysentery, or of non-specific forms of intestinal ulceration, is often met with in phthisical patients ; in whom there is no tubercle in any part of the bowel except the ileum, and where therefore it may be a question whether the ulceration originated directly in the breaking down of tubercle, or whether, as seems most likely, it took its origin in simple excoriation caused by the constant passage of irritating secretions from the tubercular bowel above, just as the mucous membrane of the trachea becomes so often excoriated in the course of pulmonary phthisis.

Many intestinal ulcers doubtless cicatrize and leave behind them no traces of their former existence, or, at most, a smooth depression with puckered edges. In other cases, however, and indeed in a large proportion of them, results of more or less serious importance follow.

Sometimes, where a vast continuous extent of surface has been destroyed, as we see occasionally in the rectum and other parts of the large intestine, the mucous membrane never does become restored ; and even in cases where the destruction of tissue has been much more limited, the ulcer may assume the character often presented by the chronic ulcer of the stomach, and be ready, as that is, to break out again and again under apparently the most trivial provocation. But generally when a large ulcer heals wholly or in part, some degree of contraction of the calibre of the bowel is the consequence,—contraction which takes place both in length and in breadth, but which from obvious causes manifests itself most conspicuously in the latter direction. Stricture, in fact, often follows such contraction, but especially, and indeed almost exclusively, when the ulceration which has given rise to it has occupied the whole circumference of the bowel, as it does often in tubercular disease, and always after the separation of a mass of invaginated bowel.

Another very common sequence of ulceration is perforation of the intestinal walls at the seat of ulceration, and the consequent communication of the interior of the bowel either with the peritoneal cavity, or with that of some hollow viscus. The most frequent of these communications is that with the peritoneum. Perforation occurs more frequently in enteric fever than in any other kind of disease, taking place generally somewhere in the lower three feet of the ileum, and rarely in the colon. It occurs occasionally only in the course of tubercular ulceration of the bowel, and then also generally in the lower part of the ileum. It is induced sometimes by the constant fretting kept

p by the pressure of some hard irritating body, such as a gall-stone
some other form of intestinal concretion. Sometimes it follows
pon the ulceration and softening of the mucous membrane, which
tend the undue distension taking place often in the bowel above
n impediment. Sometimes, again, it results from the separation of
eshly united surfaces, as in intussusception. And indeed it may
appen in the course of any form of ulceration, or weakness, whether
ependent on mere thinning, or softening, or ulceration, or gangrene.
he actual perforation, at least so far as regards the peritoneum,
hich is always the last part to yield, is due generally, perhaps
lways, to laceration. And although the result of the lesion is
eneral and, with few exceptions, rapidly fatal peritonitis, the lips
f the perforation and the contiguous portion of bowel are almost
lways found adherent by lymph to some neighbouring viscus.
ndeed perforation into the peritoneum is sometimes staved off, or
holly prevented, by the previous occurrence of localised adhesive
eritonitis. It is by the intervention of such adhesion that a per-
orating ulcer of the bowel comes usually to communicate with some
eighbouring hollow viscus. The ulcer, having first eaten its way
hrough the thickness of the parietes of the bowel, next perforates
he layer of adhesions, then the walls of the attached viscus; and
hus establishes a more or less free passage between them, and
ermits a more or less ready interchange of contents. Sometimes an
bscess-like cavity lies between the two organs which communicate,
nd forms the medium of their communication. Such communica-
ions, though generally perhaps permanent, are not always so; and
heir closure is effected usually by the retreat of the bowel from
he organ to which it is adherent, and the consequent formation of
a hollow funnel-like passage between them, which becoming longer
and narrower finally closes at its narrowest end, or that furthest from
the bowel. There are probably none of the abdominal viscera
between which and the bowels communication may not be established
by means of ulceration beginning on the side of the bowel. Thus,
not infrequently, contiguous portions of the small intestine are found
opening into one another, or small intestine into the transverse or
some other part of the colon: and thus the rectum or sigmoid flexure,
or even the small intestine, may be found to communicate with an
ovary or with the urinary bladder; or the duodenum, and perhaps the
transverse colon with the gall-bladder; or the stomach with the trans-
verse colon; or again almost any part of the intestinal canal may open
through the abdominal parietes, forming a fæcal fistula, or artificial
anus. In some cases the perforating ulceration begins in a diver-
ticulum of the ileum, or in one of the false diverticula occurring
sometimes in the large intestine. Mr. Sydney Jones[1] records a case in
which ulceration of a false diverticulum in the sigmoid flexure led
to a passage between that part of the bowel and the bladder. The
results of some of these communications are perhaps of little im-

[1] Path. Trans. vol. x.

portance; other communications, however, are not only of dang
consequence but also of much interest. Among these latter an
cially communications between the colon and the stomach or
denum, which lead to the occasional or constant vomiting of a
fæces, and the escape of undigested food into the large intestine;
communications with the urinary bladder, which occasion the a
of flatus and of fæces into that viscus, with other consequences
are easy to foresee.

(*b*) *Ulceration beginning from without.*—Ulceration. of the b
beginning from without occurs generally in connexion with
abscess of which the intestine has been made to form a p
of the parietes. The abscess is sometimes distinctly p
sometimes occupies a viscus which becomes adherent to the b
at the point where perforation is about to take place. So
the purulent matter infiltrates the cellular tissue of the m
or of some other peritoneal duplicature, and thus reach
intestinal walls. If the external abscess attacks a p
bowel covered with peritoneum, it generally causes the a
of that membrane in the first instance to a comparatively
extent: then the matter undermines it, and accumulates b
it and the muscular coat; soon the muscular coat becomes
softened, and perforated in one or more spots, when again a
cumulation of matter takes place between the muscular a
mucous membranes, which latter then forms a larger or s
hemispherical bulging towards the interior of the bowel, a
convexity of which ulceration soon ensues, and the comm
tion between the abscess and the bowel is completed. Or a
hollow viscus may open by ulceration into the bowel, having
caused adhesion, exactly in the same way that the bowel open
other organs. By the processes here indicated, peritoneal ab
discharge themselves into various parts of the bowel; inflamed o
tumours communicate with the rectum, sigmoid flexure, or other p
an ulcerated gall-bladder, or an abscess of the liver perforate
duodenum or transverse colon; an abscess of the kidney or o
form of retro-peritoneal abscess opens on the one side int
ascending colon and cæcum, on the other into the descending c
or, by burrowing beneath the peritoneum, reaches the rectum,
perforates that. In a similar way, too, an abscess of the liver, or c
an empyema, may empty itself into the cæcum or some other p
the large intestine, in or just above the pelvis.

In a few instances, tubercular deposits commencing at the p
toneal surface gradually invade the whole thickness of the bo
forming here and there large knots of tubercular infiltration of
intestinal walls, which gradually softening lead to the ulceration
the mucous surface over them, to the formation of a tubero
abscess, and even to a communication between the interior of
bowel and the cavity of the abdomen.

It may, perhaps, be added here, that malignant disease of the bo

not only causes ulceration of the mucous surface, but not infrequently produces perforation into the abdomen, and is, perhaps, the most frequent cause of complex and unusual communications between neighbouring cavities, and these and the external surface.

II. SYMPTOMS.—*The symptoms* which ulceration of the bowels produces are so constantly associated with the symptoms of those morbid states of system on which the ulceration depends, and are so frequently mixed up with symptoms due to the various complications which follow upon ulceration, that we have seldom the opportunity of studying them in their simple form; and, indeed, if we omit all reference to the symptoms of its complications, we leave very little to be said upon the symptomatology of ulceration. It may be stated generally, that ulceration of the bowels is attended in the first instance with more or less marked febrile symptoms, which assume, if the disease become chronic, a distinctly and indeed typical hectic character; that the affected bowel is more or less tender on pressure, a character which is especially observable if the ulceration be extensive, or if it occupy the cæcum and other parts of the large intestine; that there is some impairment of nutrition marked by emaciation and debility, and feebleness of circulation; and that there is, above all, something abnormal in the action of the bowels and in the evacuations. The stools in ulceration of the bowels are generally liquid, contain an abnormal quantity of the fluid secretions of the bowel, and not infrequently more or less blood; they are, moreover, often pea-souplike in colour and consistence, and much more fœtid than in health; further, they are usually passed much more frequently than natural, and the patient suffers from frequent colicky pains and from tenesmus. But all these symptoms are liable to much modification, and one or even all of them may be absent. Thus, sometimes ulceration is present, especially if it occur high up in the small intestine, without occasioning any obvious disturbance of the bowels. I recollect very well the case of a man who died from gradually increasing emaciation and debility, with no symptoms sufficiently characteristic to point to any one organ as the seat of the disease, and in whom after death the only visible lesion was pretty extensive chronic ulceration at the upper part of the ileum. The bowels, indeed, may be constipated from first to last, as we now and then observe in cases of enteric fever, and as happened in a case of extensive ulceration of the large intestine which I have quoted in another article, and in which death, and probably the ulceration itself, were due to simple constipation. Ulceration of the larger bowel is much more constantly associated with the passage of frequent and thin evacuations than is ulceration of the small intestine: these may be purely diarrhœal when the upper part of the large intestine is alone involved, but assume a more and more decidedly dysenteric character in proportion as the ulceration affects its lower part; in which latter condition the evacuations, though frequent and passed with extreme tenesmus, are scanty, mucous, and often sanguinolent, and occasionally only containing a little true fæcal matter. It is

in this dysenteric form of disease, moreover, that the evacuations become most offensive, the fœtor being sometimes, even though no gangrene be present, putrid and almost insufferable. Besides the slight oozing of blood which tinges the evacuations in diarrhœa of a dysenteric character, hæmorrhage to a considerable amount sometimes takes place, hæmorrhage which may be continuous or recurrent, and sufficient in quantity to destroy life. This accident is not very infrequent either in enteric fever or dysentery, and occasionally results from the perforation of a comparatively large vein or artery. There is little to add, even in regard to the diarrhœa which attends tubercular disease of the bowels, excepting that as the intestinal disease is mostly a progressive one, the diarrhœal symptoms, having once declared themselves, tend to become progressively more and more severe, and that it is for the most part in those cases of phthisis which are attended with intestinal complication that the emaciation is most rapid and becomes most extreme. This is not the place to discuss the various symptoms which are caused by stricture, and by perforation of the bowel, and by the communication of the bowel with other organs, nor to enter upon the description of those symptoms which attend typhoid or dysenteric ulceration.

III. Treatment.—*The treatment* of ulceration merges in the treatment of the various diseases with which it is connected, and admits, indeed, of but little independent remark. But putting all its complications out of the question, our aim in the treatment of ulceration would seem to be, first, to promote the healing of the ulcer, and to prevent, as far as possible, the local mischances which are apt to follow ; second, to check the abdominal discomfort and the diarrhœa which so rapidly weaken the patient ; and third, to support his strength directly by all means at our disposal. Whether there are any medicines which are capable of being made to act directly on an ulcer seated at a distance from either outlet may be a matter of doubt; still, from our knowledge of what drugs are useful in ulcers of the stomach and of the lower end of the large intestine, we are justified at least in hoping that some benefit, however infinitesimal, may result from the employment of the same medicines in the treatment of the deeper-seated disease. On these grounds, bismuth, nitrate of silver, iron, copper, the mineral acids and other remedies, have been frequently employed, and often with apparent benefit. But rest, which is so useful an adjunct in the treatment of so many diseases, is of inestimable value in the treatment of ulceration of the bowels. The violent and frequent peristaltic movements and writhings which the ulcers themselves give rise to, tend obviously to prevent them from healing, and add greatly to the danger of perforation; purgative medicines should therefore be entirely, or at least as much as possible, avoided, and further, the exalted peristaltic movements which attend the disease should be restrained. For this purpose various astringent medicines may be used,—lime, tannic acid, chalk, and vegetable astringents ; but far more useful than these, as a rule, is opium, in one

or other of its various preparations. There are probably few simple combinations more generally useful than the aromatic powder of chalk and opium, and the compound kino powder. But it is well to bear in mind that opium cannot always be taken in these cases. Chronic ulceration of the bowels is often attended with an irritable condition of the mucous membrane of the mouth and stomach, manifested by dryness, soreness, and, perhaps, cracking of the tongue, and heat at the stomach, with nausea—conditions which the use of opium unfortunately often intensifies. If opium then cannot be administered, astringent medicines with carminatives must be alone employed; or some other form of sedative, such as hyoscyamus, belladonna, Indian hemp, hydrocyanic acid, &c. must be resorted to. Opium may often be given with advantage in the form of suppository or of enema. It need scarcely be added that it is never desirable by these means to produce prolonged constipation; and that to obviate this contingency, either the medicines which have produced it must be left off or given in diminished doses, or simple enemata must be employed. It is obvious that the various measures which have just been enumerated, while they check peristalsis, act with equal efficacy in fulfilling the second indication of treatment,—namely, the arrest of diarrhœa. Our third and last object, the maintenance of the patient's strength, must be attained by the exhibition of tonic medicines, and the careful administration of food and stimulants. The form of tonic to be given must obviously be made to accord with the treatment selected to restrain peristalsis and diarrhœa; it must also be adapted to the condition of the patient, as regards his general health and his digestive functions. In the same way the diet must be regulated: nothing should be permitted which is known to disagree with the patient; everything should be well cooked, well masticated, and easy of digestion, and food should be given in moderate quantities, and at regular if not frequent intervals. Farinaceous foods are in many cases most suitable, but eggs, fish, and fowl may often be used with great advantage. Butchers' meat is sometimes wholly inadmissible. For stimulants, nothing, perhaps, is better, in a general way, than brandy and water, sherry, or madeira.

For reasons which are sufficiently apparent, and which have indeed been already indicated, the remarks on the treatment of ulceration are intentionally meagre, and point rather to general principles than to details.

CANCEROUS AND OTHER GROWTHS OF THE INTESTINES.

BY JOHN SYER BRISTOWE, M.D., F.R.C.P.

(1) *Cancerous disease,* to any serious extent, much more rarely affects the intestines than the stomach, and the small intestine much more rarely than the large. Of all parts of the intestinal canal, the rectum seems to be the most frequently thus affected, the sigmoid flexure next. Yet the bowels are very often the seat of a trivial amount of cancerous deposit; for peritoneal cancer, which is a not uncommon form of disease, is almost always attended with more or less involvement of their serous surface. Cancer rarely originates in the substance of the intestinal walls; but involves them by extension from the serous membrane, from the mesenteric and other abdominal lymphatic glands, from the connective tissue of the lesser omentum, venter ilei, or pelvis, or from the stomach, or the pelvic genito-urinary organs, especially the uterus and vagina. When commencing from the peritoneum it makes its appearance in that membrane, in the form of lenticular tubercular elevations, which tend to increase in number and to enlarge, and then to coalesce, so as to form a tolerably smooth or somewhat nodulated lamina of various thickness. Generally the cancerous deposits appear first, and are most abundant, in the vicinity of the lines along which the peritoneum leaves the bowel; and whether the disease begins in the peritoneum or in the substance of the mesentery and similar processes (but especially in the latter case), the subserous connective tissue becomes largely infiltrated and thickened, and the bowel firmly fixed to it or set as it were in it. It is naturally in the loose tissues around the lower part of the rectum, the cæcum, and the duodenum, that the development of subperitoneal cancer is most abundant; and sometimes these parts are thus reduced to mere channels, excavated, as it were, in the substance of a solid mass. Cancerous disease of the outer surface of the bowel may be almost universal, or it may affect tracts of bowel of various lengths; or, again, a band of cancerous deposit may encircle the bowel at some point (generally in this case, the lower part of the large intestine), while many or a few isolated cancerous nodules are scattered at distant intervals over other parts of the peritoneum.

Cancer beginning on the outer surface tends no doubt soon

invade the tissues internal to it; but although there is cer-
a great tendency in it to spread laterally, it is remarkable how
tly, even in extensive peritoneal cancer, the muscular and
coats escape. When the disease extends inwards, growths of
continuous with those placed externally, perforate the mus-
oat, which generally becomes at the same time increased in
ss and marked with vertical bands, of which some appear to
ply fibrous. Subsequently the disease invades the submucous
in which it spreads both laterally and vertically, forming a
or less well-defined, rounded, or nodulated tumour, at first
the mucous membrane which is still moveable over it, then
ng that membrane, and rendering it smooth and fixed. At
age nodules of cancer, having no apparent continuity with pre-
g cancerous masses, are apt to appear in the substance of the
membrane. Then soon ulceration takes place, which is some-
receded by the formation of a kind of false membrane on the
d surface, and is often attended with more or less sloughing of
cerous mass. The diseased tract thus becomes excavated, and
resents either a hard, smooth, cupped surface, or one in which
granulations are intermixed with sloughing hollows; the edges
hickened, and either callous and tolerably smooth or sprouting
h cancerous excrescences.

direct ill-effects of cancer of the bowels are various. In some
specially when the mucous membrane is involved in some con-
le area, diarrhœa of a more or less uncontrollable character
utes to hasten the patient's death; in other cases, and generally
he large intestine is the seat of disease, and a limited portion
el only is involved, stricture takes place; in other cases, serious
l hæmorrhage arises, either from the general surface of an ulcer,
onsequence of the erosion of some large vessel in the progress
ulceration; and in other cases, again, the bowel opens into the
eum, and extravasation of its contents and peritonitis ensue, or
mications take place between it and other portions of bowel, or
rgans, giving rise to special symptoms of more or less urgency
nger.

different kinds of cancer affect the bowels in much the same
tion as they affect the stomach; and present, as they do in the
rgan, certain specific peculiarities which may be briefly adverted
irrhus tends to produce contraction of the parts which it
s, and is especially that form of cancer which causes stricture.
cer which it yields is very often smooth and excavated; but
mes, when scirrhus extends from the outer part of the bowel to
cous membrane, it assumes in the latter situation the character
cancer, and forms there projecting growths, or an ulcer with a
cy to sprout. Encephaloid cancer presents various degrees of
and vascularity, and rarely causes obstruction of the bowel,
by the formation of a tumour, or series of tumours, springing
s mucous aspect and projecting into its cavity. The tumours

are rounded, or lobulated or even villous, and have a great tendency
to ulcerate or slough, and bleed. The melanotic variety of ence-
phaloid rarely affects the intestines except secondarily, and in the
form of minute discrete black spots, scattered for the most part
over the peritoneal surface. Epithelial cancer occasionally involves
the rectum by spreading to it from the uterus and vagina; and
occasionally, also, arises independently in the lower part of that tube.
I am not, however, aware that it ever originates, or is indeed found,
in other parts of the intestinal canal. Colloid cancer, or (if it be
preferred) colloid disease, affects the bowel usually like scirrhus and
encephaloid, from the peritoneal surface, and gradually like them
extending through the intestinal walls spreads pretty widely in the
substance of the mucous membrane, at the surface of which it appears
in the form of groups of minute vesicles, reminding one of patches
of herpes or of eczema, or (if the fibroid element be in excess) in the
form of whitish wheals not unlike those of scirrhus. These become
eroded, or more or less excavated, but remain pretty smooth, and
secrete in abundance the transparent glairy fluid, with which the
interstices of colloid material are filled. Colloid cancer comparatively
rarely involves the mucous membrane of the bowel, at any rate to
a serious extent. It sometimes appears in the cæcum, sigmoid
flexure, or rectum, as a primary disease. Mr. W. Adams[1] records a
case in which a colloid tumour, as large as the fist, springing from
the posterior part of the rectum, projected into it, and caused symptoms
of stricture.

It is difficult, if not impossible, to discuss the symptoms and treat-
ment of intestinal cancer apart from the symptoms and treatment of
abdominal cancer generally, or from those of cancer of the stomach
and rectum, or from those of its chief local consequences,—namely,
obstruction and perforation; it is, moreover, needless, for these are
all considered at length in other articles.

(2) *Fibroid infiltration and thickening*, identical with the fibroid
form of so-called "scirrhous" pylorus, is met with occasionally in
the bowels, where also it constitutes one form of "scirrhus." Its
chief, perhaps only, seats are the sigmoid flexure and rectum, where it
produces results resembling in almost every particular those which
have been described as belonging to true scirrhus. It seems, however,
to differ from that in its purely local character, in the absence of all
secondary deposits, as well as in its elementary constitution.

(3) *Villous growths* are of occasional occurrence in the large
intestine, particularly in the sigmoid flexure and rectum. They
generally occupy a limited and well-defined area, which sometimes
amounts to three or four square inches or more, and sometimes encircles
the gut. The portion of the parietes corresponding to the villous
surface is always infiltrated and thickened to a greater or less degree
with a kind of fibroid material, which forms the basis from which the
villous excrescences spring. The mucous coat and submucous tissue

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[1] Path. Soc. Trans. vol. i.

e the parts principally thus affected, and sometimes indeed grow it into a tumour with a constricted neck. The villi are abundant and close-set, easily distinguishable, especially if the tumour be bated in water, often of considerable length, conical, cylindrical or lub-shaped, and branching. As we have already seen, villous outgrowths are sometimes distinctly cancerous; but certainly most of those which have been met with in the large intestine seem clearly to have been of a benign character. The presence of a villous tumour sometimes causes hæmorrhage from the bowels or dysenteric diarrhœa; but its ultimate tendency seems always to produce obstruction. In most of the recorded cases death has been the result of stricture. Occasionally, when the growth is situated but a short distance from the anus, it admits of removal by operation.

(4) *Polypi*, or outgrowths of a non-malignant character, are not very infrequently discovered *post mortem* attached to the intestinal mucous membrane, especially to that of the lower part of the ileum, ascending colon, and rectum, and are sometimes present here in vast numbers. They seem generally to resemble ordinary cutaneous fibro-cellular or molluscous tumours, and consist, like them, of an outgrowth of connective tissue invested in a layer of mucous membrane, which still for the most part presents its normal structure. It seems not improbable that they occasionally originate in connexion with the edges of ulcerated patches; but they doubtless more frequently arise independently of any discoverable pre-existing cause. In an early stage they form mere rounded bead-like excrescences, looking like enlarged solitary glands; but they soon elongate, and generally at the same time increase in some degree in other dimensions. When thoroughly developed, they form for the most part cylindrical outgrowths from about a quarter of an inch to an inch in length, and from the thickness of a probe up to that of a director, with extremities which are sometimes bulbous and cauliflower-like, and then highly vascular, and tending to bleed. Sometimes they occur in groups of two or three, or two or three spring from the same pedicle. In the lower part of the ileum, similar bodies, but of a flatter and more leaf-like character, appear occasionally to be produced by mere elongation of portions of valvulæ conniventes. The polypi which have just been described are, as far as I know, of little or no consequence; they occur in persons of all ages and of both sexes, and do not seem to cause any symptoms. Solitary polypi, however, sometimes attain a large size, and may then produce great inconvenience, if not more serious mischief. Pedunculated fibro-cellular polypi from any size up to that of a small pear are now and then met with in the ileum, and are supposed to occasionally cause intussusception; their most common seat, however, is the rectum, in which situation they cause irritation of the bowels, tenesmus, more or less copious bleeding, and other discomforts. These solitary tumours are generally pretty smooth, but are sometimes lobulated or even warty, and mostly abundantly vascular on the surface.

(5) *Other growths* in the intestinal walls are of no practical importance; they are rare, are not productive of symptoms, and do not therefore call for description. Among them may be enumerated circumscribed submucous deposits of fat; small cysts in the same situation; erectile tumours (Rokitansky[1] considers the polypi above described as being erectile) ; and glandular tumours (in two cases[2] I have met with tumours in the small intestine which resembled the pancreas accurately in structure). Lastly, it may be mentioned that calcareous matter is sometimes deposited in small masses, either on the peritoneal or mucous surface, or in the substance of the intestinal walls.

[1] Path. Anat. Syd. Soc. Trans. vol. ii.
[2] Dr. Montgomery, Path. Soc. Trans. vol. xii. p. 130.

ISEASES OF THE CÆCUM AND APPENDIX VERMIFORMIS.

BY JOHN SYER BRISTOWE, M.D., F.R.C.P.

æcum and its appendix are liable, in a greater or less degree,
those affections which have been described as incidental to the
nal canal generally. But while some occur here comparatively
, or are of trivial consequence when they do occur, others
g partly to the connexions and position of the organs, partly to
capacity and shape, and partly to their structural peculiarities)
e them with exceptional frequency, or induce results which are
teristic either in their gravity or in some of the other features
they present.

NERAL ACCOUNT OF DISEASES OF THE CÆCUM AND APPENDIX.

lammation in its simpler forms affects the cæcum at least as fre-
ly as it affects any other part of the gastro-intestinal mucous
rane. Dysenteric inflammation is only less common here than
n the rectum and sigmoid flexure. Ulceration of a non-specific
is perhaps more often met with in the cæcum than in any other
d tract of bowel. The ulceration of enteric fever is always more
sive and more advanced in the cæcum than in the colon or
m, and occurs in it about half as frequently as it occurs in the
. Tubercular disease, which affects the large and small intestine
equal frequency, is also generally more severe in the cæcum
in other parts of the large intestine. Cancerous diseases are not
uncommon in this part. And again the degenerative results of
ic inflammation, and of lardaceous and other forms of deposit,
polypoid growths, occur equally in the cæcum and in the colon and
part of the ileum. The ileo-cæcal valve and vermiform appen-
re for the most part involved whenever the cæcum is the subject
y of the morbid processes which have just been enumerated.
margins of the valve are indeed not infrequently destroyed by
ation. And the appendix especially rarely fails to present more
s ulceration when typhoid or tubercular deposits occur in other
of the large intestine.

Strictures of the cæcum form (according to Dr. Brinton) 4 per cent. of fatal strictures of the large intestine. Some degree of contraction at this part is, however, a good deal more common than these figures would seem to indicate. The causes of contraction are, cancerous or other deposit or growth in the walls, and the cicatrization which follows ulceration, especially tubercular and dysenteric ulceration. Dilatation of the cæcum occurs casually, as dilatation occurs in other parts of the intestinal tract, from the temporary accumulation of fæcal matters, or flatus, or both. And it occurs also, as in other situations, as a result of obstructive disease in some part of the bowel below it. In this case the dilatation may become very great; and according to circumstances the parietes may be thinned or hypertrophied. It is a point of some importance that not infrequently, even when obstruction is pretty low down, the cæcum is more largely dilated than the length of bowel between it and the seat of obstruction.

Perforation of the cæcum is far from uncommon. Sometimes this ensues on long-continued distension, either from thinning, softening, and sudden laceration, or from the ulceration which so frequently attends distension. Sometimes it is caused by simple perforating ulcer, or, by the irritation of some foreign body which has been swallowed, has traversed the small intestine safely, and has become arrested in the cæcal pouch. Sometimes it occurs in the course of dysentery, enteric fever, and tuberculosis. Sometimes it is a result of cancerous ulceration. And sometimes it depends on diseases outside the bowel, such, for example, as cancer occupying the venter ilei, or the extension of a psoas, renal, hepatic, pleural, or other abscess. Perforation may take place directly into the peritoneum, lighting up fatal peritonitis; or it may establish a communication between the cavity of the bowel and the sub-serous cellular tissue of the venter ilei, or some adjoining part, and lead to the formation of a fæcal abscess; or again it may cause a communication with some adherent coil of small intestine.

We can scarcely speak of stricture of the appendix vermiformis; yet occasionally, as a result of ulcerative destruction of the mucous membrane or of other morbid processes, the whole organ becomes shrivelled up or atrophied. Dilatation, too, sometimes occurs when its orifice is obliterated or obstructed. Then the appendix becomes elongated and plump (perhaps as thick as the little finger), presents often false diverticula (resembling on a small scale those of a sacculated bladder), and is distended with a glairy transparent fluid, the secretion of the mucous membrane. Again, the appendix is apt to become perforated. This accident may be caused in any of the several ways in which the cæcum itself becomes perforated. It occurs sometimes perhaps as a result of mere ordinary ulceration. Dr. Murchison[1] records a case in which it happened in the course of typhoid fever, but where there was no escape of fæcal matter. Leudet[2] states that out of thirteen

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[1] Path. Trans. vol. xvii. p. 127.
[2] Archiv. Gen. Aug. and Sept. 1859, and New Sydenham Society's Year Book for 1860.

ases of perforation of the appendix, which he observed, six were due
o tuberculosis. This statement, however, is certainly not in accordance
with general observation. The usual cause indeed of perforation is
undoubtedly the presence of some concretion which, by fretting the
surface with which it is in contact, excites ulceration, to which the
perforation is consecutive. Fæces habitually find an entrance into the
appendix; but their entrance and escape constitute a normal process,
on which as a rule no ill consequences supervene. Together with the
fæces, however, insoluble bodies of small size—seeds, bristles, pins,
pieces of bone, shot—are apt to enter the appendix; and some of these,
from their pointed or angular form, or from their size, become retained
and cause ulceration. Perforation has been caused by bristles, by pins,
and by pieces of bone: and indeed it was formerly generally believed
that the foreign bodies causing perforation were all of external origin,
and for the most part cherry or date stones, or stones of a similar cha-
racter. There seems no doubt, however, that bodies of this bulk rarely
find their way into the appendix, and that what have been mistaken for
them have been concretions resembling them somewhat in size and
shape, but differing from them in origin and in constitution. The
concretions generally met with vary from perhaps the size of a small
pea to that of a date-stone: they are sometimes of waxy consistence
and lustre throughout; sometimes brownish, for the most part fæcal,
and laminated; sometimes again composed almost entirely of earthy
phosphates; they consist obviously of the admixture, in unequal
proportions, of ordinary fæcal matters and of the secretions from the
mucous membrane of the appendix, and have obviously formed in
the situation in which they are found, either round a nucleus of solid
matter which has been first precipitated and concreted there, or round
some comparatively small body of extraneous origin. Sometimes two
or three of these concretions are present at the same time. Perfora-
tion of the appendix occurs at any part, sometimes at or near its base,
sometimes at its point or within half an inch of it, sometimes again
in some intermediate spot. The resulting orifice varies in shape and
size. Perforation may take place directly into the peritoneal cavity,
causing generally acute and rapidly fatal peritonitis, sometimes a
circumscribed peritoneal abscess; or actual perforation may be pre-
ceded by adhesion of the appendix to neighbouring parts, and the
formation of a limited abscess either among the adhesions or in the
surrounding structures.

It may be added here, in order to complete our summary of diseases
incidental to the cæcum and appendix: that the most common
form of intussusception, and the most frequent in children, is that
in which the cæcum is engaged; that the cæcum is occasionally
the subject of internal strangulation, and that more frequently its
appendage takes part in the production of strangulation of other
parts of the intestine: and lastly, that the cæcum and its ap-
pendage, together or separately, are not very infrequently contained
in an ordinary hernial sac.

II. Ulceration and Perforation of the Cæcum and Vermiform
Appendix.

(*a*) *Pathology.*—The terms "Typhlitis" and "Perityphlitis,"—the
former signifying inflammation of the walls of the cæcum, the latter in-
flammation in the tissues surrounding the cæcum,—are used frequently,
though somewhat vaguely and indiscriminately; but I believe are
generally applied to those cases in which there is perforative ulceration
either of the cæcum or of its appendix, and in which, therefore, there
is either limited suppuration in the neighbourhood of these parts, or
sudden peritonitis. The perforation in the great majority of cases, no
doubt, occurs in the appendix vermiformis: sometimes, however, it
occurs in the cæcum itself, beginning there generally from ulceration of
the mucous membrane, but occasionally from an abscess situated
upon its outer surface. The results which ensue have already been
briefly enumerated.

In some instances the ulcer perforates that portion of the bowel
which corresponds to the mesenteric attachment, or, if occurring
elsewhere in the bowel, the area in which perforation is about
to take place becomes adherent to some viscus in the vicinity,
or to some portion of the parietes of the true or false pelvis. The
morbid process may stop at that point; or the escape of fæcal matter
and flatus into and among the tissues may lead to the formation of
an abscess, with more or less surrounding inflammation and indura-
tion. In the latter event the abscess usually enlarges pretty rapidly,
and in enlarging takes a course dependent more or less on its
original position, in one case descending into the pelvis, and opening
perhaps into the rectum, in another passing out with the pyriformis
muscle and presenting in or below the buttock, in another form-
ing a lump in the groin immediately above Poupart's ligament, or
passing along the inguinal canal towards the scrotum, or along the
psoas and iliacus muscles into the upper part of the thigh. But
indeed when once an abscess has formed, although it may tend as a
rule to elect one of several courses, there is scarcely any conceivable
direction which under certain circumstances it may not take. No
doubt it generally presents itself in the groin as a hardness or lump
superficial to the position which the cæcum normally occupies. An
abscess of this kind may empty itself and become healed by discharg-
ing its contents either through the orifice in the cæcum which gave
rise to it, or through an opening at any one of the spots at which,
as has been shown, it may present; or having burrowed largely
it may form a sinus or series of sinuses which never become
obliterated. The communication between the abscess and the cæcum
is sometimes maintained, at other times is more or less speedily
obliterated.

In other cases the bowel ruptures directly into the peritoneum, ex

citing at once acute peritoneal inflammation. This may be so severe as almost directly to prove fatal: but in most cases the patient survives sufficiently long to allow of the more or less complete obliteration by adhesion of the general cavity of the peritoneum, and the formation in the vicinity of the perforated bowel of a circumscribed peritoneal abscess. It is not improbable that in some cases the perityphlic abscesses, the course and progress of which have been already discussed, are really peritoneal abscesses. And it may be added that the abscesses originally unconnected with the peritoneum not infrequently open suddenly into it and evoke, as does the sudden rupture into it of the cæcum or of its appendix, sudden and severe inflammation there.

The statistics of "Typhlitis," using this term as expressive of all the morbid conditions which have just been described, are not very easy to obtain. But as regards the statistics of that section of typhlitis which relates to perforation of the cæcal appendage followed by fatal results, they seem to show very conclusively that this accident occurs chiefly in early life, and much more frequently in males than in females. Thus, in ten cases analysed by Bamberger,[1] eight were males, two females; eight were below thirty years of age, two above thirty. In thirty-two cases collected by Dr. Crisp,[2] twenty-nine were males, three females; five were under ten years, thirteen between ten and twenty, seven between twenty and forty, and seven between forty and sixty. And in eight cases recorded in the "Pathological Transactions" since the publication therein of Dr. Crisp's paper, five were males, three females; and their ages ranged from thirteen to thirty-four.

The duration of typhlitis must obviously be very various. When the perforation takes place directly into the peritoneum, death for the most part ensues speedily—generally indeed in from three days to a week; life may, however, even in this case be prolonged, in consequence of the formation of a circumscribed peritoneal abscess, to two or three weeks or more, and it is not impossible that under the latter condition recovery sometimes takes place. In seven of Bamberger's cases the duration of the illness varied between twenty and fifty days. But when a fæcal abscess forms in the tissues in the neighbourhood of the cæcum no definite limits can possibly be assigned to the duration of the case; sometimes the patient recovers pretty speedily; sometimes, the case, having got apparently into a chronic state, proves suddenly fatal with symptoms of peritonitis; sometimes again the patient lingers for months, or even years, with a constantly discharging abscess or a succession of abscesses.

(δ) *Symptoms.*—The symptoms which attend and indicate typhlitis are mainly either those of acute peritonitis, or those of local suppuration, or a complex of both. In those cases in which sudden rupture

[1] Ueber die Perforation des wurmformigen Anhangs. : Schmidt's Jahrb. 1859, vol. ci. p. 184.
[2] Path. Trans. vol. x. p. 151.

takes place into the peritoneum, there are very often no prem
symptoms whatever; occasionally, however, some localised un
or pain, due to the ulceration which is taking place, or to some
mation of the peritoneal surface corresponding to the seat of ulc
precedes for a longer or shorter time the violent outbrea
patient, while in the enjoyment apparently of perfectly good
and at the moment probably of making some muscular ef
attacked with sudden acute pain in the region of the cæcum, f
speedily by collapse, and the diffusion of pain and tenderne
the whole extent of the abdomen. The symptoms in fact o
peritonitis are almost instantaneously set up, symptoms whic
differ from those of idiopathic peritonitis in the suddenness o
invasion and the severity of the collapse, and differ in no degre
those which attend rupture of the bowel from other causes, r
of the stomach, or rupture of the bladder. It is needless to d
the character of the abdominal pain and tenderness, and on the
nitic condition of abdomen which ensues, on the dorsal dec
which the patient is generally compelled to assume, on the qu
and shallowness of his respiratory acts, on his feebleness of
shrunken and anxious expression, and for the most part fr
vomitings and hiccough. But it may be observed, that in spit
rather perhaps in consequence of, the unbearableness of his pa
patient sometimes assumes positions and makes contortions of h
which might seem to be incompatible with the presence o
peritonitis; that sometimes the peritonitic indications remain
strictly limited to the neighbourhood in which they commence
that very frequently indeed they do not extend above the line
by the transverse colon; and that sometimes as the case pr
proceeds even towards its fatal issue, general peritonitic sym
almost entirely subside, leaving perhaps a distinct fulnes
dulness and tenderness, due to the formation of a circums
abscess, in or about the right lumbar or iliac, or the hypo
region.

In those cases in which an abscess forms in the neighbourh
the cæcum, there are in the first instance pain and tenderness
region of the cæcum, together with rigors and other general sym
of inflammatory fever. Generally, too, there is some distinct
and tenderness to be felt. The symptoms indeed are for the
part those which might be caused by suppuration, of whatever
occupying the venter of the ileum. When the abscess extends
wards into the pelvis, or remains deep-seated, the case is na
obscure. When, however, it tends to point anteriorly we find t
ness and hardness become gradually more and more pronounce
fulness in fact grows into a more or less distinctly hemispherical
over which the integuments become œdematous and congested.
times, even at this stage, the swelling gradually subsides and
pears, owing to the abscess having discharged itself into the
but more frequently it still enlarges and ultimately opens exte

charging a greater or less amount of fœtid pus, sometimes having distinct fæcal odour, or even obviously containing fæcal matter and bubbles of gas. It must, however, be remembered, that not infrequently the communication with the bowel has been cut off before the abscess opens externally, and that the absence of ordure or of gas does not necessarily show that the abscess has not commenced in perforation of the bowel. Sometimes the abscess, after having discharged itself externally, gradually fills up, and complete and permanent recovery takes place. Sometimes, after it has healed externally and appears to have been cured, it forms afresh and presents in the same, or some other, situation. In other cases it remains as a permanently open fistula, or as an artificial anus. In these latter cases symptoms of hectic come on, the patient becomes thinner and feebler, and though in some cases life may be prolonged for a considerable period, death generally ensues from gradual exhaustion at the end of a few months, at the outside a year or two.

There are, however, many cases in which the perforation of the bowel causes abscess in the first instance, and peritonitis subsequently, either in consequence of a fresh intestinal perforation, or of a rupture of the abscess into the peritoneum, or of the mere extension of inflammation by contiguity. These are the cases in which, for the most part, perforation of the cæcal appendix is said to be preceded by premonitory symptoms; and there can be no doubt that it is chiefly by taking these into consideration that cases of perforation of the appendix are estimated by Bamberger and others to have a duration much longer than we know belongs to mere peritonitis the result of perforation.

It might naturally be supposed that any disease, affecting so important a part of the alimentary canal as the cæcum, would be attended with some disturbance of the functions of that canal. It does not appear, however, that there is any constant disturbance. Sickness is very often entirely absent. Constipation is mentioned as having been present in many cases at or about the time of perforation; but there does not seem to be any definite connection between these two conditions. And diarrhœa not uncommonly supervenes in the course of the disease; but this again would seem to be for the most part a mere incidental phenomenon.

There are many diseases, or incidents of disease, with which typhlitis may be confounded. It may be worth while briefly to call attention to some of the more important of them. Acute peritonitis of idiopathic origin may sometimes, from its suddenness and severity, and from its happening to take the lower part of the abdomen as its starting point, be thought to have its origin in perforation of the appendix. So also may the peritonitis caused by perforation of the bowel in enteric fever, especially in those cases in which the febrile symptoms are slight and the patient is not compelled to give up work until the sudden rupture takes place. The same also may be said of all those cases in which peritonitis arises from the perforation

of a hollow viscus, or of an hydatid or other abscess, from the laceration of the cyst of a tubarian or ovarian pregnancy, or from the extension of inflammation from various pelvic organs, especially those of the female. Again, the local suppuration which attends many cases of typhlitis may in some one or other of its stages be easily confounded with abscesses of other kinds, which form in, or find their way into, the region of the cæcum; among which may be enumerated, psoas abscesses, and abscesses extending from the kidney, the spinal canal, and the pleura. It may similarly be confounded with ovarian tumours or inflammation, with cancerous tumours of the venter ilei or glands in the vicinity of the cæcum, and even under some circumstances with aneurismal tumours.

(c) *Treatment.*—The treatment of typhlitis may be dismissed in a few words, not because it is unimportant, but because it resolves itself into the treatment of enteritis and the treatment of a localised suppuration: the former of which has been discussed elsewhere in this volume; the latter of which is mainly a surgical question. As regards those cases in which there is a direct communication between the bowel and the peritoneum, our main reliance must be placed upon opium; which must be administered, partly with the object of relieving pain, partly with the object of restraining intestinal movements and preventing further escape of fæcal matters. For similar reasons, all purgative medicines must be most carefully avoided. In reference to the employment of local measures, such as leeching, fomentation, and the like, no special observations need be made. It is most important of course to administer nourishment and stimulants; and owing to the comparative absence of vomiting, their administration by the mouth can for the most part be much more readily carried out than in cases of enteritis or of obstruction. It is, however, at the same time essential that the bowels should not be overloaded, and therefore that the food which is thus given should be nutritious, capable of easy digestion and absorption, and given in small quantities at frequent intervals. But here indeed, as in many other cases of stomach and bowel disease, it is important to consider how far we may supplement or replace the duties of the stomach and smaller intestine in the absorption of nutriment, by the regular employment of nutritious enemata. When we have to deal with a case of inflammation, circumscribed in the situation of the cæcum, it need scarcely be said that leeching, poulticing, fomentation, and other local remedial measures will naturally be called into requisition; and that, so soon as there are clear indications of the presence of pus, an opening should be made for its evacuation; and that the abscess having been once opened should if possible be kept open, until we have evidence that its deeper parts or ramifications have become healed. In cases of this kind also the use of opium, though not so universally imperative as where there is peritonitis, is generally desirable if not indispensable; and in them also, purgatives, though not perhaps to be absolutely prohibited should be employed exceptionally only, and with the greatest caution,

. there can be little doubt that if constipation be sufficiently
: to call for medical relief, relief will be afforded best, and by
safely, by the use of enemata. Lastly, in these cases, as in
where there is abundant and long-continued suppuration and
; is of paramount importance that the patient should be sus-
ɤ abundance of nutritious food, that he should have habitually
:oportion of stimulus, and that the use of tonic medicines,
ɤ vegetable bitters, and tonic treatment generally, should be
ically enforced.

COLIC.

BY J. WARBURTON BEGBIE, M.D., F.R.C.P.E.

THE term Colic is derived from the Greek Κῶλον, the colon, or large intestine.

DEFINITION.—The essential character of Colic, as ordinarily understood, is severe pain in the abdomen (in a restricted view, in the colon), augmenting for a time in severity, and then gradually subsiding; occurring in paroxysms, not stationary, but, on the contrary, moving from place to place, accompanied by a sense of constriction and tearing, for the most part also by that of expulsion.

The term Colic is now used in nearly the same way as the ancient writers employed that of Κωλικός. It is, however, abundantly evident that the disease described under that name, by Aretæus for example, was of a much more serious nature than ordinary colic; it was indeed a frequently fatal disorder. In treating of Colics, Περὶ Κωλικῶν, the learned Cappadocian physician remarks: "Κωλικοὶ δὴ κτείνονται εἰλεῷ καὶ στρόφῳ ὀξέως." By Linnæus, among the early nosologists, Colic is placed in the class "Dolorosi," and is thus defined: "Intestini dolor umbilicalis cum torminibus." Vogel, using a similar expression to denote the class, explains the disease as follows: "Dolores: Colica, dolor spasticus intestinorum cum obstipatione, nausea, et vomitu." Sauvage more simply and briefly styles Colic "Dolor intestinorum;" and Cullen, correctly assigning the disease a position in the class "Neuroses," of his nosological system, of which "Spasmi" is the third order, has thus described it: "Dolor abdominis, præcipue circa umbilicum torquens; vomitus; alvus adstricta." By French and German writers the terms "Colique" and "Die Kolik" are respectively employed when treating of this disease.

A vast variety of painful spasmodic affections has been described under the name of Colic. Of these it may only be necessary to adduce as illustrations the following: "Colica Hepatica," "Colica Nephritica," "Colica Uterina," as applied to spasmodic pain, sudden in its occurrence, and apparently affecting the liver, kidneys, or uterus. These expressions are eminently faulty, and it is desirable that their use should be entirely abandoned.

It is to the consideration of the true or simple Colic, the "Colica spasmodica" of not a few writers, that the present article will be

voted. "Lead Colic," or "Colica Pictonum," and for which many
her synonyms have been employed, will be separately considered,
hile the occurrence of Colic, or of colicky pains, as a symptom of
ifferent abdominal affections, inflammatory and otherwise, will be
oticed in the descriptions of these maladies themselves.

SYMPTOMATOLOGY OF COLIC.—As has already been stated in the
definition of Colic, pain is its essential and most characteristic feature.
This pain is seldom continued or uniform for any length of time, but,
on the contrary, is marked by the occurrence of remissions or inter-
missions, and likewise by exacerbations, which are frequently of very
great, even intense severity. So extreme is the pain of Colic at times
as to cause persons of heroism to utter loud groans and cries. While
the whole abdomen or any part of it may be the seat of suffering, the
peculiar twisting pain is specially experienced in the situation of the
umbilicus, as Cullen observed : " præcipue circa umbilicum torquens." [1]
Great restlessness and frequent turning of the body, changing from
place to place, distinguish the sufferer from Colic. He does not rest
in bed, but is prone to rise and pace up and down the room ; bending
forwards, he presses his hands over the belly; and when the pain aug-
ments in severity is glad to fling himself on his face on the bed or
sofa. Usually, while the pain lasts, the trunk is flexed, the upper part
bent forward over the lower. If the patient be in bed and lying on
the back, the lower limbs with bent knees are often brought in con-
tact with the abdominal parietes and are thus retained for some time
by his hands. A position of this kind is meant when French writers,
in reference to the sufferer from Colic, use the expression, "le malade
pelotonne," the patient rolls himself into a ball. By very firm
pressure over the abdomen, as by lying on the belly, the pain is some-
times mitigated or even for a time removed, and this circumstance is
of some importance in distinguishing a spasmodic from an inflamma-
ry pain, in so far as the latter is invariably aggravated by pressure.

The form of the abdomen is altered during the continuance of Colic.
There may be, and this condition is fully the more frequent, distension,
with which there is associated the development of flatus on a large
scale, or the parietes of the abdomen may, on the other hand, be re-
tracted. The condition of a distended colon, the seat of pain, may be
mistaken for that of gastric distension and pain. When the former,
however, occurs as a phenomenon of the attack of Colic, there are
present also other indications of intestinal suffering, such as irregular
contractions which may frequently be felt by the hand or seen, bor-
orygmi, and specially the sense of bearing down towards, and con-

[1] A recent, perhaps the most recent, French writer on Colic (M. Martineau), in describ-
ing the pain, remarks : " La douleur est toute spéciale. Les malades en proie à une
olique éprouvent une douleur vive, exacerbante, mobile, ayant une grande tendance à
l'irradier. Elle se traduit par une sensation de constriction, de resserrement, de tortille-
ment, ou par une sensation de déchirure et même d'éxpulsion."—*Nouveau Dictionnaire
de Médecine et de Chirurgie pratique*, vol. viii.

striction at, the anus.   Besides, as Dr. Wilson Fox [1] has pointed
pain arising from the large intestines is seldom felt so much at
ensiform cartilage (the common seat of gastric uneasiness) as in
right or left hypochondriac regions, while there exists a distinct
ence between the notes to be elicited on percussion, from the
organs; that from a distended colon being the less prolonged,
having a higher pitch.

Great general depression is capable of being produced by an attack
Colic. This is seen in the frequently pale countenance of the suf-
whose pulse also is found to be extremely feeble, while the surface of
body is bedewed with a cold and clammy perspiration.   The rela-
of constipation to Colic is most important.   A confined condition
the bowels is usually, though not invariably, as some writers
asserted, associated with Colic; and not unfrequently, when the b
have been efficiently acted on by medicine, the pain, which may
been of the severest type, entirely disappears.   Neither is this
however, the constant result, for, notwithstanding the operation
laxative and cathartic remedies, the pain in some instances
persistent.   Such cases are infinitely less alarming than those in
obstruction of the bowels continues, while the abdominal pain
diminishes or disappears, for in these circumstances the occur
sooner or later, of a regular attack of ileus is to be apprehended;
in the former case, the free movement of the bowels, although
immediately, and it may be not even speedily, bringing relief to
fering, is surely succeeded by such before any lengthened period
passed.   In some instances of Colic, a confined condition of the bo
is really the cause of the attack of painful spasm, while in other
constipation is the effect of the spasm.   In the more protracted
of Colic, a general febrile state is liable to be induced.   Vomiting
accompany Colic, but is by no means a constant or characteristic sy
tom of this disorder.   Much importance is to be attached to the p
in Colic, for by its condition we are not unfrequently able to dis
guish between a simple, although severe spasmodic affection, and
inflammatory disorder.   It is to be remembered, moreover, that
some circumstances the latter is not unapt to supervene upon
former.   Now, in Colic, while the suffering is even intense, the p
may be little if at all altered.   Assuredly it is by no means uncom
to find the pulse, under such circumstances, remaining tranquil
in fact altogether normal.   Smallness of the pulse, associated
marked depression of the circulation generally, hardness and irr
larity, are, on the other hand, of sufficiently frequent occurrence in c
of Colic.[2]   The respiration is hurried, and frequently unequal.
voice is apt to be affected in cases of marked severity; it beco
hoarse, while at times it is so enfeebled as to be almost obli
The accession of Colic is by no means uniform or exact.   The

[1] The Diagnosis and Treatment of the Varieties of Dyspepsia, p. 53.
[2] In describing the pulse of Colic, Henoch remarks, "Der Puls ist klein und
lich." (Klinik der Unterleibs-Krankheiten.)

be established suddenly, even abruptly, and without any appa-
ause, or it may come on gradually, succeeding the occurrence, for
e longer or shorter, of abdominal uneasiness, and very probably
casional cramps, which are clearly traceable to some sufficient
. Not less variable are the progress and duration of the malady.
y exist for days, or last only for hours, or even minutes. These
larities are largely determined by the precise causes of the
ks. An irregular intermittence is a characteristic feature of
; the duration of the painful seizures, and of the intervals which
ate them, being subject to great variety.

THOLOGY OF COLIC.—Although the relation of the abdominal pain
spasm in Colic to nerve irritation, is obscure, the following remarks
ar to be called for. It has been clearly shown by carefully con-
ed experiments, and is now admitted, that the pneumogastric
es possess an influence on the movements of the intestinal canal.
experiments as those referred to, have exhibited the contractions
e muscular coats of the intestines under the application of elec-
l irritation to the vagi, of as rapid and violent a character as those
luntary muscles, when their motor nerves have been subjected to
ilar irritation. Again, when on irritating the ganglionic plexuses
unding the aorta, by means of the rotary apparatus (*durch den*
orischen Apparat), the small intestines and colon, which had been
iously wholly inactive, when the current began to operate were
d with universally active movements, which continued for a long
after the current was interrupted. It is of further interest to
that among central portions of the nervous system it is the medulla
gata, which when irritated by the galvanic current excites in a
ed manner the movements of the stomach and the intestinal
. Budge saw the same result produced in rabbits, but in a less
e, by irritation of the cerebellum. The spinal cord and cerebrum
ss no such influence. All experimenters have described the
ments of the intestinal canal as distinctly peristaltic or vermi-
[1] M. Martineau, in his interesting article on Colic to which re-
ce has been made, has pointed out that while the pneumogastric
is more especially distributed, as is well known, to the stomach
iver, a portion of the right nerve passes to the semi-lunar ganglia
astomose with the splanchnic nerves of the great sympathetic,
thus to form the solar plexus. Galvanization of the solar plexus
f the superior mesenteric ganglia equally causes contraction of the
intestine and more rarely of the large. Valentin has made the
important observation that an irritation of the fifth nerve, at the
of the skull, invariably gives rise to peristaltic movements of
mall intestine, especially of the duodenum and upper part of the
um. Such being proved experimentally, we can understand the
rence of intestinal spasm or Colic, as the direct consequence
me forms of cerebral irritation. And although, as Romberg has

Romberg, Lehrbuch der Nervenkrankheiten des Menschen ; Darmkrampf.

remarked, little is known respecting the influence which is exerted by the affections of the spinal cord and brain, upon spasms of the bowels, the very potent operation of the emotions, fear and fright especially, but in some instances also joy, in increasing the movements of the intestines is thoroughly appreciated.

ETIOLOGY OF COLIC.—Certain temperaments appear to predispose to the occurrence of Colic. Of these the nervous and lymphatic are the most distinguished. Sedentary occupations act in the same manner. The influence of age and sex is sufficiently marked to be worthy of notice. In youth and adult age, Colic is more common than in advanced life, and among females it occurs more frequently than among males. Among the exciting causes of Colic, one of the most frequent is the presence of some indigestible article of food in the bowels. The influence of cold in producing attacks of Colic is also remarkable, and particularly, it has been noticed, cold applied to the feet. There are some individuals who are certain to suffer from an attack of Colic, if by any means their feet have become cold. The association of biliary derangement with the occurrence of intestinal spasm is not uncommon, and this particular form of the disease has been designated "Bilious Colic." Its distinctive features are the vomiting of biliary matters, and the presence of a more or less icteric tint of the conjunctivæ and surface of the body. Lastly, under this head, it is to be held in remembrance that in some instances the existence of a gouty or rheumatic habit of body plays a decided part in the origination of attacks of Colic, although it may probably be admitted that such constitutional disorders are still more potent in determining the true enteralgia or neuralgia of the bowels, a disease which is to be distinguished from Colic.

TREATMENT OF COLIC.—To relieve pain, and generally speaking to act on the confined bowels, are the chief indications for treatment in Colic. In the milder instances of the disease, unaccompanied by any notable derangement of the "primæ viæ," this can usually be accomplished by the external application of warmth, or of rubefacients, such as mustard and turpentine, and by the administration of a little stimulant, or carminative mixture. A small quantity of brandy with hot water, a teaspoonful of the compound tincture of cardamoms in warm water, or twenty drops of the compound tincture of chloroform, will be found very serviceable for this purpose. Preparations of peppermint, ginger, and cloves may also be similarly employed. In more severe cases of Colic, or in instances where the remedies already mentioned have failed to relieve the pain, it will be necessary to administer anodyne medicines, and as early as possible to evacuate the bowels. The preparations of opium are most useful among the former; the compound tincture of camphor or English paregoric—in doses of thirty to sixty minims—or a full dose of laudanum. With these a dose of castor oil, or compound rhubarb powder (Gregory'

nixture), should be given, and repeated after a short interval if relief to pain and solution of the bowels be not obtained.

A tablespoonful of castor oil with twenty-five drops of laudanum in peppermint water, or two teaspoonfuls of Gregory's mixture, with a teaspoonful of compound tincture of camphor, and a similar quantity of aromatic spirit of ammonia in a small wineglassful of cinnamon water, will be found most available prescriptions in such cases.

When the attack of Colic has speedily succeeded the taking of some indigestible article of food, it may be advisable to produce vomiting by the administration of an emetic of ipecacuanha wine, or by draughts of hot water.

Should the bowels not respond to the mild remedies already mentioned, it will be necessary to have recourse to the use of stronger cathartics. Of these, sulphate of magnesia, particularly with the addition of a little sulphuric acid, as Henry's salts, and senna, also the compound jalap powder and calomel, may be regarded as the chief.

The employment of laxative enemata should also be had recourse to. A large injection of warm water will frequently be found most useful in relieving the pain, and in effectually acting on the bowels in cases of Colic.

The prophylactic treatment of Colic consists in a careful regulation of diet, particularly in the avoidance of all indigestible articles of food, and in the protection of the surface of the body from the injurious influence of cold. Wearing flannel over the abdomen, and the warm covering of the feet, are specially to be enjoined.

COLITIS.

By J. Warburton Begbie, M.D., F.R.C.P.E.

There seems to be some ground, at all events, for supposing that the large intestine may be the seat of inflammatory action, differing in essential particulars from the dysenteric process which will be immediately described. To indicate the simple inflammation of the colon, as distinguished from dysentery, the term *Colitis* has been employed. *Colonitis* has been used in the same sense. The French have the word *Colite*, and the Germans the expression *Entzundung des Schleimhautes des Kolons.*

In dysentery the mucous membrane of the rectum and colon is primarily involved, while the pathological changes which are so eminently characteristic of the disease are wrought in it. In Colitis, on the other hand, there is in all probability a commencement of inflammation in the submucous or connective tissue, which underlies the mucous membrane, the glandular structures of the latter being in the first instance uninvolved. The result, however, is a diffuse gangrenous inflammation of the mucous membrane; and when this has occurred, there is no possibility of distinguishing the ulceration thus formed from that which has resulted from the dysenteric process.

It is, however, to be borne in mind that the most experienced physicians and ablest writers have differed in respect to the essential pathology and the characteristic morbid appearances in dysentery. The necessary existence of ulceration has, for example, been denied by some, and the special participation of the glandular structures of the colon, so commonly conceived to hold true of dysentery, has been equally opposed by others. In these circumstances it must be admitted that great difficulty at present exists in the way of correctly distinguishing between the different forms—if there really be different forms—of inflammatory disease affecting the colon, and renewed investigation with careful examination of the various structures and tissues entering into the anatomy of that portion of the intestine, is required before any satisfactory conclusions on the subject can be arrived at.

DYSENTERY.

By J. Warburton Begbie, M.D., F.R.C.P.E.

DEFINITION.—A febrile disease, in which inflammation affecting the glandular structures of the large intestine chiefly—although sometimes extending to the small—and producing ulceration, tends to terminate in sloughing of the mucous membrane. The disease is accompanied by much nervous depression, and is characterised by tormina—severe pains in the abdomen of a griping nature—followed by frequent scanty and bloody stools, straining, and tenesmus.

The term Dysentery is derived from two Greek words—δυς, hard or bad, and ἔντερον, a piece of the guts, intestines. Δυσεντερία was itself employed by Hippocrates and other Greek writers to signify a bowel complaint, or bloody flux.

SYNONYMS.—Tormina; Tormina intestinorum; Fluxus dysentericus; Fluxus cruentus; Fluxus torminosus; Rheuma ventris; Febris dysenterica; Colonitis; Bloody Flux; Dysenteria; Flux de Sang (French); Die Ruhe, Die rothe Ruhe (German); Dissenteria (Italian); Dysenteria (Spanish).

HISTORY.—Dysentery has been known as a disease since the earliest period of medical history. In several of the Hippocratic treatises, but especially in the following,—Περὶ ἀέρων, ὑδάτων, καὶ τόπων, Προγνωστικόν, and Ἀφορισμοί,—are many interesting remarks regarding the symptoms and treatment of Dysentery, also the prognosis to be founded upon it, to be met with. Aretæus has described the Dysentery with his usual conciseness, and even more than his usual ability. In Cælius Aurelianus, but still more in Celsus, much information may be found regarding Dysentery, as the disease was known in the days of these celebrated Latin writers. Coming down to modern times, Sydenham, Ramazzini, Morton, Huxham, Cleghorn, Morgagni, Zimmerman, and Sir John Pringle (in his celebrated treatise on Diseases of the Army), are among the more distinguished of the numerous writers on Dysentery.[1]

[1] For a full and instructive account of the history and geographical distribution of Dysentery, see Hirsch, "Handbuch der historisch geographischen Pathologie," article "Ruhe," vol. ii, p. 194.

Dysentery is placed by Cullen in class first, "Pyrexiæ," and of it the fifth order, "Profluvia." Of the latter his definition is, "Pyrexia cum excretione aucta naturaliter, non sanguinea." Dysentery, Cullen defines as follows: "Pyrexia contagiosa; dejectiones frequentes, mucosæ, vel sanguinolentæ, retentis plerumque fæcibus alvinis; tormina; tenesmus."[1]

It is customary to distinguish between acute and chronic Dysentery, also between epidemic and non-epidemic or sporadic Dysentery. To the non-epidemic disease we are now to direct attention—the epidemic Dysentery having been already considered by Dr. Maclean in Vol. I.

SYMPTOMATOLOGY. — The essential characters of Dysentery are severe pains of a griping nature in the belly, followed by frequent and bloody stools, defecation being accompanied by much straining and tenesmus. The later symptoms are the most characteristic. Watch a patient affected by Dysentery at stool: he sits a long time, straining; his features are distorted by the pain he suffers; the discharge from the bowels may be, often indeed is, but scanty: still he sits. The strong desire to remain at stool, accompanied by griping and straining, is expressed in the word tenesmus. Scarcely can such patients at times be persuaded to leave the stool and return to bed, until they feel so faint as to be unable longer to maintain the sitting posture, and sometimes while on the stool they faint.

Straining and tenesmus do not occur in diarrhœa, they are peculiar to dysentery; and so also are the other symptoms, named in Cullen's definition; the passage of blood and mucus, the fæces being for the most part retained, or after a time passed in the form of small, often hard, scybala.

Acute Dysentery.—The disease in this form may occur without any premonitory symptoms; more commonly, however, it is preceded by such. General uneasiness, lassitude, impaired appetite, disagreeable sensations in the abdomen, confined bowels, or a loose condition of the bowels, are among the more frequent of the premonitory symptoms. These may have existed for a few days, when a chill is experienced, or sometimes a chill or rigor is the very earliest indication of departure from a healthy state. To these succeed the febrile symptoms, heat of skin and quickness of the pulse. Much variety exists in respect to the degree of the general or constitutional disturbance which accompanies the local affection in Dysentery. That may be very slight indeed; the disease may even run its course without fever. On the other hand, the constitutional disorder may be severe, and is not unfrequently profound, assuming an adynamic or typhoid character. In the simpler variety of the disease, there are at the commencement griping pains in the belly, those pains to which the name of "tormina" is now generally applied. This term was

[1] Synopsis Nosologiæ Methodicæ, p. 308.

ιt used by Celsus.[1] "Proxima," he says, "his inter intestinorum
la *tormina* esse consueverunt: δυσεντερία Græcè vocatur." The
mina are felt in different parts of the belly, and, like the pain of
ic, yield at one time, to return again, perhaps more severely than
ore. With the tormina there occur discharges, usually slight, from
ι bowels, and by these a partial relief to the pain is experienced.
the tormina and diarrhœa succeeds the tenesmus; and this term
y be understood as including the frequent desire to go to stool, and
ι reluctance to leave it, with the very distressing feeling of bearing
wn, and burning sensation in the rectum. In every marked case of
ιsentery the tenesmus is a prominent as it is the most distressing
nptom. The discharge from the bowels affords little relief when
ι tenesmus is great. The calls to stool of course vary greatly in fre-
ιncy: in some instances they are almost incessant. Occurring in
ldren particularly, but occasionally also in adults, as a consequence
the frequent evacuations, and the tenesmus by which they are accom-
ιied, is prolapsus of the anus, which in itself requires careful manage-
nt, and may become a very troublesome sequela of the disease.[2]
Γhe discharges from the bowels in Dysentery are peculiar and
ιracteristic. At first they are usually feculent, if not entirely, at
ιst chiefly so; but very soon, becoming very scanty in amount, they
ι found to be composed of mucus, or of mucus mixed with blood,
d sometimes of nearly pure blood. When the inflammation of the
wels has advanced to a certain stage, it is common to notice the
ιearance of vitiated bile in the stools, and likewise of shreddy-
king portions of fibrine or false membrane. The odour of the
ιcuations in Dysentery is one *sui generis*, quite peculiar, and
ιhly offensive. Not unfrequently there is sympathetic irritation
the bladder, and a frequent as well as difficult micturition. While
ι chief part of the pain in Dysentery is experienced during the
ιvement of the bowels, it is not limited to that time—pain is
ιsent in the abdomen generally, aggravated by pressure. When, in
lition to the tenderness over the left side of the belly, correspond-
ι to the position of the sigmoid flexure, there is pain felt over the
gastrium and down the right side, it may be conjectured that the
ease has implicated the large intestine in its entire extent, and
not limited, as happens in milder instances, to the rectum and
ιcending portion of the colon.
More or less of fever accompanies Dysentery. In mild cases the

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Liber iv. ch. xv. The description of the disease given by Celsus is so accurate as to
rit perusal; the earlier sentences may be quoted. "Intus intestina exulcerantur; ex
cruor manat, isque modo cum stercore aliquo semper liquido, modo cum quibusdam
ιsi mucosis excernitur; interdum simul quædam carnosa descendunt; frequens deji-
ndi cupiditas, dolorque in ano est; cum eodem dolore exiguum aliquid emittitur;
ιue eo quoque tormentum intenditur; idque post tempus aliquod levatur; exigua
ιies est; somnus interpellatur; febricula oritur; longoque tempore id malum, cum
ιeteraverit aut, tollit hominem aut, etiamsi finitur, excruciat."
[1] "Durch die heftigen Anstrengungen wird auch nicht selten, zumal bei Kindern, ein
ιlapsus ani herbeigeführt, der sich entweder von selbst wieder zurückzieht oder reponirt
rden muss."—HENOCH, *Klinik der Unterleibs-Krankheiten*, Ruhe, Band 3, p. 235.

feverish disturbance, as already stated, is slight, but, on the other hand, in the more decided instances of the disease, the constitutional disturbance is evidenced by the quickness of the pulse, the augmented temperature of the surface, scanty secretion of urine, and the coated condition of the tongue. In the milder cases of Dysentery there is no special implication of the nervous system; the pulse in such, although frequent, is full and of good strength: neither nausea nor vomiting, except of occasional occurrence, are present; and although the local malady may be severe, the disease wears throughout a sthenic character. But it is not always so; an asthenic or adynamic form of Dysentery also occurs, characterised by a frequent, small, and feeble pulse, pallor and coolness, rather than warmth of the skin, the occurrence of a clammy moisture over it, anxious expression of the countenance, sunken eyes, dryness and glazing of the tongue, suppression of the voice, hiccough, delirium, prominence of the abdomen, and rapid sinking. With these indications there is unusual violence in the local symptoms, particularly as regards the frequency of the discharges from the bowels. These ultimately become exceedingly offensive and watery. They present the appearance of water in which raw flesh has been washed, and are known by the name of "lotura carnium." The disease may thus prove fatal in a few days. Dr. Wood speaks of such cases as very rare, and only seen during epidemics.[1] The latter observation is no doubt correct, but only to a certain extent, for these instances of rapidly fatal dysentery, although more common in the epidemic prevalence of the disease, are occasionally met with in the non-epidemic malady. It has occurred to the writer to witness one or two very rapidly fatal cases of Dysentery, in which a remarkable depression of the nervous system was evident from the very commencement of the disease. In the ordinary form Dysentery tends to a favourable termination, and usually before the lapse of a week or eight days there are indications of amendment. The acute disease sometimes terminates in chronic dysentery.

Chronic Dysentery is characterised by the frequency of the evacuations, which, at the same time, are usually very scanty. As in the acute affection, so in the chronic, the discharges are attended by local suffering and tenesmus. Mucus, or mucus mixed with blood, sometimes with purulent matter, constitutes the bulk of the evacuations; feculent stools occur when the disease, instead of implicating the entire colon, is limited to the rectum, or involves with it only the descending portion of the former. Chronic dysentery may last for months or years. In some instances it appears to produce wonderfully little influence on the general health and strength of the invalid, but as a general rule the sufferer from chronic dysentery is emaciated, pale, and weakly; and the disease is not unapt to prove fatal, through the exhaustion consequent upon its long continuance, or owing to the establishment of a state of continual or hectic fever.

[1] A Treatise on the Practice of Medicine. By George B. Wood, M.D. Vol. i. p. 625.

the morbid conditions which are connected with, or result
cks of Dysentery, whether acute or chronic, affections of the
upy a chief place, and to these attention will be called, in
of the pathological anatomy of the disease. Anæmia, more
narked, results from Dysentery. The writer remembers to
n a case of anæmia of a very typical character, in which
d impoverishment was due to a long-continued attack of
y. To the occurrence of paralysis in conjunction with
y, Romberg has called attention,[1] and he quotes a passage
old dissertation by Fabricius: "De paralysi brachii unius et
erius lateris dysentericis familiari,"[2] in verification of the
J. P. Frank refers to the same occurrence;[3] and although
has not specially mentioned Dysentery as a form of intestinal
iving rise to a reflex paraplegia, he has emphatically done
erence to Enteritis. By Zimmerman[5] and Joseph Frank[6]
is made to paralysis of the arms and legs occurring after
y.

D ANATOMY.—As Dysentery is essentially a disease of the
estines, it is in the colon and rectum that we look for
bid appearances characteristic of its occurrence.[7] The
membrane in these portions either presents the appear-
having been diffusely inflamed, being everywhere much
, thickened, and at parts ulcerated, or, with the absence
e inflammation, there exists remarkable prominence of the
lands and mucous follicles. There exist three separate and
orms of ulceration affecting the mucous surface of the in-
-the tubercular, the typhoid, that met with in enteric fever;
dysenteric. Apart from other characteristic differences in
ctions, the last-mentioned is nearly limited in its occurrence
ge bowel, while the two former are especially met with in the
estines, and particularly in the ileum. The size of dysenteric
ries. They are sometimes small, and present a nearly cir-
m, or they are larger, irregular in shape, have an abrupt

bei der Dysenterie," remarks Romberg, "ist das Vorkommen der Paralyse
worden."— *Lehrbuch der Nervenkrankheiten des Menschen: Spinale Läh-*

ationes ad Morborum Historiam et Curationem facientes quas collegit, edidit,
t Albe rtus Haller.Tomus primus, p. 97.
um verò ad gradum doloris in abdomine vehementia apud hos vel illos evehitur,
n minus ac in colica saturnina brachii aut pedis unius vel alterius paralysis
—*De Curandis Hominum Morbis.* Auctore Joanne Petro Frank, Liber **v.** De
pars ii. p. 497.
Lectures, Edition 1864, in one vol. p. 415.
r Ruhe unter dem Volke in Jahr 1765.
Medicæ Universæ Præcepta. Auctore Josepho Frank. De Paralysi.
acous membrane of the colon, says Rokitansky, is the seat of the dysenteric
d we may state it as a rule, that its intensity increases from the cæcal valve
, and consequently is met with in the most fully-developed state in the
ture and in the rectum. It not unfrequently passes beyond the cæcal valve
ileum, but is here only seen in its mildest form.

border, are covered by a dark-coloured slough, and appear as if formed by the coalescence of several smaller ulcers. It is not uncommon to find considerable portions of more or less dense lymph, coating the reddened and thickened mucous surface. Portions of false membrane having precisely the same appearance are sometimes passed at stool; but these, while still adherent to the bowel, do not when removed usually disclose an ulcerated surface. A truly sphacelated condition of the mucous membrane is occasionally met with, and pieces of gangrenous mucous membrane, sometimes of considerable size, have been passed in the evacuations in certain cases of Dysentery. Perforation of the bowel, which is of no uncommon occurrence in the progress of typhoid ulceration, and occasionally takes place in tubercular disease of the bowels, is very rarely indeed met with in Dysentery: the mucous, submucous, and muscular coats of the colon suffer in this disease, but the peritoneal covering is not so apt to be involved. The mesenteric glands in Dysentery are frequently found tumefied and presenting a dark-bluish colour. They may be softened, but are very rarely indeed the seat of suppuration. Even when much enlarged they have not been distinguished by the presence of any peculiar morbid product such as occurs in the typhoid and tubercular tumefactions of these glands. Rokitansky describes the dysenteric process as divisible into four natural degrees or forms.[1] The anatomical characters of the *first* or lowest form are, swelling, injection, and reddening, softening (red and bleeding), serous exudation in the shape of a delicate vesicular eruption, and consequent branny desquamation of the epithelium (the latter appearance probably led Linnæus to term Dysentery "Scabies intestinorum interna"). In the *second* form, a larger surface of the bowel is involved, but still presenting a greater development at one part than another—there is copious infiltration of the submucous cellular tissue, giving rise to a greater or less number of prominences, which correspond to those parts of the mucous membrane at which the morbid process is most conspicuous. The intestine is generally in a state of passive dilatation, distended by gas, and occupied by a dirty-brown fluid, composed of intestinal secretions, epithelium, lymph, blood, and fæces. The coats of the bowel are thickened, and the submucous tissue especially in a state of tumefaction. In the *third* stage, the prominences are more thickly set, and the result is an uneven lobulated appearance. The mucous membrane investing these prominences is in part converted into a slough, or it may have disappeared, so as to expose the infiltrated submucous cellular tissue to which the remnants of the mucous membrane remain attached, in the shape of solitary dark-red, flaccid, and bleeding vascular tufts, or dilated follicles which are capable of easy removal. The contents the intestine are now of a dirty-brown or reddish, ichorous, fetid, flocculent and grumous character. In the *fourth* and highest degree, the mucous membrane has degenerated into a black, friable, carbonified mass, portions of which may be subsequently voided in the shape

[1] A Manual of Pathological Anatomy. Sydenham Society's Edition, vol. ii. p. 83.

ibular laminæ (so-called mortification of the mucous membrane). he submucous cellular tissue appears to be infiltrated with sero-anguinolent fluid, or dark blood; or it is pale, and the blood contained n its vessels is converted into a black solid mass. Purulent infiltra-ion of the submucous tissue is also found. The affected portion of he bowel, which contains a putrid fluid resembling coffee-grounds, may be either in a state of passive dilatation, or (and this is more requently the case) collapsed. In the higher degrees of the dysenteric rocess the muscular coat of the colon suffers; its tissue becomes ondensed, pale, ashy, and friable. In the same degrees, the peritoneal overing does not completely escape, it presents a dirty-grey discolora-ion, has lost its lustre, and here and there dilatation and injection of s capillary vessels is visible, while occasionally it is covered by a thin rownish ichorous exudation. These characters afford the means of cognising the existence of an advanced stage of Dysentery, while as et the intestine has been unopened, and the mucous surface unexposed. okitansky has some very interesting observations on the termination f Dysentery. Provided disorganization of the mucous membrane has ot occurred, a cure results through the return of normal cohesion, nd the generation of a new layer under the desquamated epithelium. n the more intense degrees of the dysenteric process, and when dis-rganization has taken place, the mucous membrane having undergone ore or less destruction, one of two results ensues—either a real cure f the loss of substance, with consolidation of the abraded portions of he intestine, follows, or the entire process assumes a low chronic rm, the specific nature of the disease is lost, and an inflammation tonic in character, with suppuration of the intestinal coat, occurs. ysentery is fatal through the more or less rapid, or more or less enetrating, destruction of tissue and coincident exhaustion. When ure results, the loss of substance having been inconsiderable, new issue is formed, and may so contract as to bring the edges of the mucous membrane into apposition with one another, while a cicatrix emains, which has the appearance of a large number of agminated warty excrescences of the mucous membrane, between which the sero-brous basis from which they proceed may be detected. On the ther hand, in those instances of the disease which have been dis-inguished by an extensive loss of substance, the approach of the dges is impossible, and the deeper layers of the tissue which takes he place of the mucous membrane are frequently condensed into brous bands, which form projections into the intestinal cavity, inter-aced with one another, and not unfrequently encroach upon the calibre of the intestine, in the form of valvular or annular folds, thus iving rise to a variety of stricture of the colon.

Reference has already been made to the participation by the liver n disease in connexion with Dysentery. Abscess of the liver has een supposed by some authorities to have an intimate relationship o the dysenteric process in the colon. Of the not unfrequent association of the two diseases there can at all events be no question.

Dr. Parkes[1] found, in twenty-five cases of Dysentery, seven
affected with hepatic abscess. In the large work of Mr. Anne
there are twenty-nine cases of abscess of the liver recorded, a
these no fewer than twenty-one, or nearly three-fourths, had ul
tion, more or less extensive, in the large intestine, while in two
cases there were appearances of constriction and contraction
were reasonably ascribed to the existence of Dysentery at some
period. Annesley regarded the Dysentery as the result of the dis
of the liver, or hepatitis. By certain writers, among whom Dr.
crombie[3] and the late Dr. William Thomson of Glasgow[4] may
mentioned, the concurrence of the two diseases has been regard
accidental. The former observes: "Dysentery is often accompa
by diseases of neighbouring organs, especially the liver, in whi
to be found, in some cases abscesses, in others, where protract
their duration, chronic induration. These are to be regard
accidental combinations, though they may considerably modify
symptoms." A third view, and one which has been popular in
country since it was ably upheld by Dr. Budd,[5] is that the infla
tion of the liver terminating in abscess is the result of pur
absorption from the dysenteric process in the colon. Many yea
Andral and Louis, apparently unsuspecting any connexion betw
hepatic abscess and ulcerated intestines, noticed the co-existen
the former with ulceration in the large intestines and in the low
of the ileum in two cases, in the lower end of the ileum alone in
case, in the stomach in four cases, in the gall-bladder in one. I
of the cases in which the stomach was affected, Andral conc
with reason that the ulcer was caused by the hepatic abscess bur
into the stomach. But excluding this observation, there res
seven out of fifteen instances of hepatic abscess, in which t
existed at the same time ulceration in some part of the exte
mucous surface which returns its blood to the portal vein. T
observations of the French pathologists were very far indeed
being singular. Thus Dr. Cheyne, of Dublin, in writing of the Dy
tery in Ireland, remarks that in the majority of his dissection
liver was apparently normal, but that in two cases he found abse
in its substance. But while the occasional intimate connexi
hepatic abscess with Dysentery, and of which Dr. Budd's theo
all probability assigns the true cause, has been determined, it
also be admitted that abscess of the liver frequently occurs in tro
countries wholly unconnected with Dysentery, not acknowledg
pyæmic origin, and not resulting from mechanical injury. Dr.
chison, of London, in his papers on the Climate and Disea

[1] Remarks on the Dysentery and Hepatitis of India, 1846.
[2] Researches into the Causes, Nature, and Treatment of the more prevalant D
India and of Warm Climates generally. By James Annesley. 2 vols. 4to. Lond
[3] Researches on the Pathology of the Intestinal Canal. 1820.
[4] Practical Treatise on the Diseases of the Liver and Biliary Passages. 1841.
[5] On Diseases of the Liver, 1845.

rmah,[1] pointed out that, in many cases, abscess of the liver met
th in tropical countries occurred independently of these three causes.
r. Morehead,[2] while admitting the occasional occurrence of hepatic
scess, according to Dr. Budd's explanations,—that is, by the trans-
ission to the liver of pus or vitiated secretion originating in an
lcerated intestinal surface,—is satisfied that, as a general proposition,
uch a view is altogether at variance with the results of clinical
search in India. Seventeen cases of hepatic abscess are detailed
y Dr. Morehead in which no intestinal ulceration existed. Frerichs,
moreover, is of the same opinion, although by no means denying that,
a certain cases, dysenteric as well as other forms of ulceration of the
owels may originate phlebitis of the coats of the portal veins, and so
duce hepatic abscess.[3] The abscess of the liver which is found in
timate connexion with Dysentery is the multiple abscess, small but
umerous collections of pus. This form of purulent deposition Dr.
Murchison has very distinctly shown to differ from the ordinary
bscess of the liver which occurs in warm climates. In the latter
se there is but one abscess which may attain a very large size, or in
few instances there may be two or three collections. Thus the
yæmic or multiple abscess, which is the common form of hepatic
ppuration in this country, is to be distinguished from the *tropical*
bscess of India and other hot climates; and while the latter may
exist with Dysentery, such connexion is wholly accidental. On the
ther hand, the multiple hepatic abscess, although by no means of
equent occurrence in India, is sometimes met with, but only, as Dr.
Murchison has pointed out, in connexion with Dysentery or some
ther source of purulent absorption. The only marked instance of
epatic abscess in connexion with Dysentery which has fallen under
he writer's immediate observation was that of a soldier in a Highland
egiment, who, while serving in India, became affected by the latter
isease, which ultimately assumed a chronic and inveterate form. He
was ordered home, and during his voyage to England the liver became
much enlarged. Greatly emaciated and reduced in strength, and
till suffering from frequent loose stools, he sank shortly after reaching
his country. Examination of the body after death revealed the
existence of a very large number of small abscesses scattered through-
ut the entire substance of the liver, the tissue of which was in
ifferent parts the seat of considerable induration.

ETIOLOGY.—Neither in its acute nor chronic form is Dysentery now
common disease of this country. The decline in the frequency of
ts occurrence has also been accompanied by a diminution in the

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[1] Edinburgh Med. and Surg. Journ. 1854.

[2] Clinical Researches on Diseases in India. 2 vols.

[3] "Eine causale Abhängigkeit der Hepatitis von Darmverschwärung ist also keineswegs
estgestellt, wenn auch die Möglichkeit nicht geläugnet werden darf, dass ausnahmsweise
nter begünstigenden Umständen dysenterische und andere Darmverschwärungen Phlebitis
er Pfortaderwurzeln und hierdurch Leberabscesse erzeugen können."—*Klinik der Leber-*
rankheiten, Zweiter Band, p. 113.

severity of its attacks. From producing a very considerable
mortality, as was the case in the seventeenth century, Dysente
occupies a very low place among the causes of death. Essen
disease of hot climates, its prevalence is, in these, observed to
to a considerable extent on meteorological changes, while in
rate climates Dysentery is emphatically an autumnal malady,
continued exposure of the body to an elevated temperature,
poses to the occurrence of Dysentery; this it does, in all prob
by an injurious operation on the mucous membrane of the wh
mentary canal leading to its increased excitability, and by
dering the function of the liver: thus exposed, the sudden red
of temperature, which so frequently takes place in the night s
our autumns, acts as a direct exciting cause of the disease.
while heat predisposes to Dysentery, cold excites it. Unwh
food has a potent action in the production of Dysentery. In th
unripe fruits, or even the ripe fruits when inordinately consume
vegetables, acid wines, and impure water, have particularly be
posed to act. There can indeed be no doubt that most of the
and some even of the severer cases of Dysentery which we me
are occasioned by a distinct error in diet, or are traceable
introduction into the alimentary canal of some substance or flu
deleterious or directly irritating nature. The not unfreque
nexion of Dysentery with ague, and their observed alternation, h
to the impression that the former disease, like the latter, acknow
an origin in malaria. That Dysentery may be produced by exh
from putrid animal and decaying vegetable substances, may p
be admitted; but the probability is that the relation of this dis
intermittent and remittent fevers, formerly insisted on, was not, s
speaking, etiological, but to be accounted for by the disordered
the portal circulation, which, occurring in ague, led indirectly
inflammatory affection of the colon. The contagious nature of
tery has been asserted by some authorities; facts are, however,
wanting to prove the communication of the disease from pe
person, in the sporadic form of the disease, with the consider
which we are occupied; and in regard to the epidemic Dysentery,
be admitted that the experience which appears at first sight to
this conclusion, admits of another and more satisfactory expla

TREATMENT.—Dysentery in its acute form demands an e
treatment; it is not a disease which can with safety be entr
the "vis naturæ medicatrix." Confinement to bed is of
importance, the very rest favouring the arrestment of the ma
much as movement of the body promotes its progress. Bloo
was formerly practised in the treatment of Dysentery, and wh
is severe, and continues unrelieved by warm applications a
facients, local blood-letting by means of leeches applied
track of the colon may still be had recourse to. The appl
a few leeches to the verge of the anus has been recomm

authors, and in the experience of the writer has appeared to be
cial.

indication of great importance in the treatment of Dysentery is
: the bowels from all irritating accumulations. This is best done
e employment of the gentler laxative medicines. Strong ca-
cs are not to be used. Castor oil has been almost universally
led as the best remedy for this purpose. Where much pain
the oil may from the first be combined with a little laudanum;
e more advanced stages of the disease it will be prudent to
ate the latter with it at every dose. The alternation of laxatives
piates in the treatment of Dysentery has been highly praised by
practitioners. "It is the practice of some physicians," writes
homas Watson, "to prescribe laxatives and opium together; but
s complaint it is better to alternate them."[1] Opium by not a few
een regarded as the "summum remedium" in this disease. It
the favourite remedy of Sydenham in meeting the formidable
atery of his generation, and it is in allusion to its efficacy that
prince of English practical physicians" rapturously exclaims—
here I cannot but break out in praise of the great God, the
of all good things, who hath granted to the human race, as
fort in their afflictions, no medicine of the value of opium, either
ard to the number of diseases that it can control, or its efficiency
tirpating them. . . . . So necessary an instrument is opium in
and of a skilful man, that medicine would be a cripple without
d whoever understands it well, will do more with it alone than
uld well hope to do from any single medicine. To know it only
means of procuring sleep, or of allaying pain, or of checking a
œa, is to know it only by halves. Like a Delphic sword, it can
ed for many purposes besides. Of cordials it is the best that has
rto been discovered in nature. I had almost said it was the only
[2] Opium may be administered either in full or in small doses,
ach of these methods has its supporters. It may be given alone,
mbined with ipecacuanha, in the form of Dover's powder. Ipe-
nha itself is again largely employed, and more especially of late
in India. We say *again* largely employed, for it is worthy of
k that ipecacuanha, originally known as a medicine about the
le of the seventeenth century, was first used as a remedy in
atery. Brought to Europe from Brazil by Piso, and some time
vards made the subject of experiment in Paris by Adrien
tius, it was long known as the "radix anti-dysenterica,"[3] the
is anti-dysentericus." Subsequently to its original employment
ance, in doses from one to three drachms, it was used in this
ry and its colonies by Sir John Pringle and other physicians, in
varying in amount, that ordinarily given being a scruple. More

ctures on the Principles and Practice of Physic, vol. ii.
dical Observations : Dysentery.
r an interesting account of the early history of ipecacuanha, see "Traité Théra-
e et de Matière Médicale," par A. Trousseau et P. Pidoux, vol. i. p. 666.

recently the names of Mr. Mortimer, Mr. Twining, Mr. Do
several other Indian surgeons, have been identified with the
of exhibiting ipecacuanha in Dysentery. The therapeutic
the remedy has been variously ascribed to its nauseant, its di
and its laxative or purgative effects. The latter was the vi
tained by the distinguished writer Sir John Pringle. Dr.
thus expresses himself in regard to it: "It is probable th
cuanha owes much of its usefulness in this disease to its acti
evacuant. It is a blood depurant of an effective kind. It a
increase the secretion of the whole alimentary canal, as well
liver and pancreas: under its use tormina and tenesmus d
and feculent evacuations are more quickly restored than by a
known remedy."[1] Dr. Morehead has always used ipecac
Dysentery from a consideration of its efficacy being due to its
action. This physician counsels the exhibition of the ipe
according to the plan of the late Mr. Twining,[2] viz. "from six
grains, combined with blue pill from five to two grains, and o
gentian from four to two grains, every third, fourth, sixth, or
hour, and to continue it steadily till amendment takes pla
proportion of the dose and the frequency of its repetiti
depend on the acuteness of the symptoms. The duration of th
ment, and the gradual diminution of the dose and of the fr
of its exhibition, must be contingent on the rapidity and per
of the amendment. It must also be kept distinctly in vi
whilst the treatment by ipecacuanha is being pursued, it
necessary—according as the state of the pulse or the u
of the abdomen on pressure may indicate the necessity—to
leeches; and also—according to the character and scantines
evacuations, and the greater or less fulness of the abdomen—
castor oil occasionally in moderate doses." The reliance
therapeutic action of ipecacuanha is most conspicuously exh
however, in the plan of its use suggested by Mr. Docker, and
by Dr. Maclean,[3] and now generally followed in India. "The
should be at once ordered to bed, and as quickly as possible
under the influence of ipecacuanha in large doses. Some i
the propriety of first giving a full dose of Battley's sedative,
of opium, or a few drops of chloroform, with the intention of
the stomach tolerant of the remedy, and restraining nause
vomiting. I believe that the sedative in some cases is usel
acts in the manner just described. On the other hand, I have
seen ipecacuanha do its work well, and with little disturbance
stomach, without opium. Should it be determined to premise O
thirty drops of the tincture should be given, and in half an
followed by from twenty-five to thirty grains of ipecacuanha,
should be given in as small a quantity of fluid as possible;

[1] Reynolds' System of Medicine, vol. i. article Dysentery.
[2] Researches on Diseases in India, vol. i. p. 560.
[3] Reynolds' System of Medicine, vol. i. p. 120.

range-peel covers the taste as well as anything else. As
ised, the patient should be kept perfectly still, and abstain
for at least three hours. If thirsty, he may suck a little
aspoonful of cold water at a time may be allowed. It is
t under this management nausea is excessive, and vomiting
ublesome, seldom setting in for at least two hours after the
as been taken. The abdomen should be covered with a
ism, or a sheet of spongio-piline sprinkled with a little tur-
er being wrung out of hot water. In from eight to ten hours,
o the urgency of the symptoms, and the effect produced by
se, ipecacuanha in a reduced dose should be repeated, with
precautions as before. All who have had opportunities of
s mode of treating Dysentery can bear testimony to the
effects that often follow the administration of one or
of ipecacuanha given in this manner. The tormina and
subside, the motions quickly become feculent, blood and
pear, and often, after profuse action of the skin, the patient
tranquil sleep and awakens refreshed. The treatment may
be continued for some days, the medicine being given in
doses, care being taken to allow a sufficient interval to
e patient taking some mild nourishment suited to the stage
se." If the writer be entitled to express an opinion regarding
a remedy which he has had but few opportunities of
in the treatment of Dysentery, but has very frequently pre-
cases of depraved action of the chylopoietic viscera, he
ed to ascribe the eminent therapeutic virtues of ipecacuanha
t action on the secerning function of the liver.
loyment of mercury in Dysentery is as warmly defended by
itioners as it is condemned by others. In all stages and
he disease Dr. Maclean deprecates its use, while Dr. Wood
t no remedial influence is more effectual in Dysentery than
rcury. Anything like the production of profuse salivation
y to be avoided; and although favourably influencing the
Dysentery in some cases, chiefly through its action on the
ill generally be admitted that in ipecacuanha, and in the
t of mild laxatives alternately with opiates, we possess
cious and certainly safer remedies.
the more chronic form of Dysentery that such powerful
us acetate of lead, sulphate of copper, sulphate of zinc,
Bael fruit, hæmatoxylon, and the sulphuric acid, are chiefly

alterative remedies copaiba and turpentine, creasote and
a, have been commended. Quinine will favourably influence
ss of malarial Dysentery, when employed as an adjunct to
dies; and iron, in the form of the pernitrate more especially,
r when fluidity of blood as evidenced by hæmorrhages and
petechiæ exist; just as in scorbutic Dysentery when chronic,
resh fruits are indispensable articles of treatment.

Enemata of warm water cautiously introduced into the rectum are frequently grateful to the patient, and are useful in the early stages of Dysentery, in bringing away hardened scybalous masses, the continuance of which in the bowels is attended by much irritation and suffering. Opiate, enemata, and those containing ipecacuanha, and various astringents, may sometimes be employed with good effects. In Dysentery assuming a typhoid or adynamic type, it is necessary to support the patient's strength by the exhibition of stimulants; but these are, as a general rule, not well borne in this disease, and should always be administered with the greatest degree of caution.

The diet in Dysentery is of much importance. When the disease is comparatively slight and unattended by serious febrile symptoms, most farinaceous foods may be allowed. When, however, the severer form of the disease is in existence, bland drinks are alone admissible: milk with lime water, or Carrara water, may be regarded as the chief article of diet, and generally speaking is the one most relished by the patient.

Great attention should be paid to preserving the cleanliness of the patient, the dress, and bed-clothes, and in keeping the atmosphere of the sick-room as pure as possible, impregnated as it must from time to time become with the offensive odour of the discharges. The use of Condy's fluid, of weak chlorine vapour, or of carbolic acid for this purpose, is invaluable.

Sponging the surface of the body with tepid or warm water is desirable, and is usually found most grateful by the patient.

It may be added in connexion with the treatment of chronic Dysentery, that change of air is frequently more efficacious than the use of drugs. Removal to the sea-coast, or a voyage, is specially to be recommended.

A flannel belt round the abdomen is an article of clothing which the convalescent from Dysentery, as well as all those who are prone to suffer from this disease, should adopt and constantly wear.

DISEASES OF THE RECTUM AND ANUS.

By Thomas Blizard Curling, F.R.S.

An acquaintance with the numerous disorders of the lower bowel is absolutely necessary to qualify the medical practitioner to form a right diagnosis and judgment of the diseases of adjacent organs, as well as of the alimentary canal. Thus, complaints of the rectum are able to be mistaken for affections of the uterus and even of the bladder; a discharge from a fistula in ano has been supposed to proceed from the vagina. Patients have been treated for obstinate diarrhœa, when the actual disease has been stricture in the lower bowel, or a lacerated perinæum and sphincter; and obstructions referred to the abdominal intestines have been discovered when too late to exist in their pelvic termination. The following is a table of the diseases of the rectum and anus; they can be treated of only very concisely in the space allotted to this subject :—Congenital Imperfections; Hæmorrhoids; Prolapsus of the Rectum; Irritable Ulcer; Irritable Sphincter; Nervous Affections of the Rectum; Villous Tumour of the Rectum; Polypus of the Rectum; Fistula; Chronic Ulceration; Stricture; Cancer; Atony; Anal Tumours and Excrescences; Prurigo Ani.

CONGENITAL IMPERFECTIONS OF THE ANUS AND RECTUM.—These may be classed as follow :—1. Imperforate anus, without deficiency of the rectum. 2. Imperforate anus, the rectum being partially or wholly deficient. 3. Anus opening into a *cul-de-sac*, the rectum being partially or wholly deficient. 4. Imperforate anus in the male, the rectum being partially or wholly deficient, the bowel communicating with the urethra or neck of the bladder. 5. Imperforate anus in the female, the rectum being partially deficient, and communicating with the vagina or uterus. 6. Imperforate anus, the rectum being partially deficient, and opening externally in an abnormal situation by a narrow outlet. 7. Narrowness of the anus. A few other congenital deviations have been observed, but they are of very rare occurrence, the seven forms enumerated above being those most commonly met with.

The classification of these imperfections is founded on states which can generally be recognised during life. Unfortunately the condition of the terminal portion of the intestinal canal, and its relations to the parts around, cannot be predicated with any certainty. In cases of

imperforate anus, or of anus opening into a *cul-de-sac*, the intestinal canal may terminate in a blind pouch at the brim of the pelvis, the rectum being wholly wanting; or an imperfect rectum may form a shut sac, descending to the floor of the pelvis, or as low as the neck of the bladder in the male, or the commencement of the vagina in the female. It is known that the anal portion of the bowel is developed distinctly from the upper portion, and that the two afterwards approximate and unite, the diaphragm or septum disappearing by interstitial absorption. A failure in this process is the cause of the second form of congenital imperfection. The cases of imperforate anus in which the rectum communicates with the urethra or vagina depend on the original existence of a cloaca, the malformation being due to an incomplete separation during fœtal life. These conditions are the result of an arrest of development at different stages. The blind pouch in which the intestinal canal terminates is sometimes connected to the anal integument, or to the anal *cul-de-sac*, by a cord prolonged from the bowel above. These cases are not, like the preceding, the result of a non-formation of the rectum, but are produced by an obliteration of the bowel which was originally well formed; the obliteration being a pathological change due probably to ulceration and adhesion which had taken place during intra-uterine life.

These imperfections of the rectum can be remedied only by operative measures which vary according to the nature of the irregularity; and this treatment unfortunately often fails in obtaining a vent for the fæces, or in securing a permanent and sufficient passage. In cases of failure in reaching the bowel at the natural site, life may still be preserved by making an artificial anus either in the left loin or in the left groin. For several reasons the latter is the best situation for the operation in infants.[1]

HÆMORRHOIDS.—The hæmorrhoidal veins distributed in the submucous tissue at the lower part of the rectum communicate in loops, and form a plexus which surrounds the bowel just within the internal sphincter. The veins are best seen when somewhat congested, their deep purple hue being very apparent through the thin mucous membrane with which they are in close contact. The plexus is then found to be about three-quarters of an inch in length, and composed of veins of various sizes, arranged for the most part lengthwise and in clusters, being especially collected in the longitudinal folds of the rectum. The plexus does not extend lower than the external sphincter, but veins branching from it pass between the fibres of the internal sphincter, and descend along the outer edge of the former muscle close to the integuments surrounding the anus.

These veins are very liable to become dilated and varicose, giving rise to the disease termed *hæmorrhoids* or *piles*. When the plexus beneath the mucous membrane is thus affected, the hæmorrhoids are said to be *internal*. When the veins beneath the integuments out-

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[1] See "Observations on the Rectum," by Mr. Curling. 3d edit. p. 221.

us, but a second may form at a subsequent period. The second
of external pile consists of flattened prolongations of skin. They
nerally the chronic results of the first form, a projecting fold
ter absorption of the coagulum having undergone further growth.
utaneous excrescence contains no clot, and no enlarged or vari-
eins; but clots and dilated veins may often be found at its base.
is sometimes only a single broad flat excrescence at the side of
us; but there are often two, one on each side, and occasionally
Similar excrescences occur as the result of irritating discharges
the bowel, and are common in stricture and ulceration of the
n.
changes in structure consequent upon internal hæmorrhoids
good deal. In general the lower veins of the hæmorrhoidal
are dilated irregularly, or into pouches, which are filled with
compact coagula. A bunch of varicose veins crowded in the
ends of the longitudinal folds produce prominent projections of
ucous membrane, and deepen the pouches between the folds.
r three of these prominences unite so as to form a transverse fold
ithin the sphincter. After a time the mucous membrane and
cous tissue become greatly hypertrophied. Thus are deve-
elongated processes of a polypus form, and projecting trans-
folds. The arteries, which are abundantly supplied to the lower
f the rectum, enlarge considerably, so that the mucous mem-
involved is not only thickened, but extremely vascular. Such
he changes found in dissection, but the description conveys
faint and incomplete impression of the condition of the parts
ed during life.
rnal piles seldom attract attention until they have become
ped so as to protrude at the anus in defecation. They then
t a remarkable diversity of appearance according to their
er, size, and condition. The protrusion may consist of only one
ized pile, found usually towards the perinæum or front of the
A single pile, consisting of a bright red projecting membrane
cted with a loose fold of integument, and readily extruded, often

forming a circular prominence. The aspect of extruded piles
much upon their condition, whether congested, inflamed, or con
by the sphincter. In a relaxed condition of the sphincter, the
softish tumours of a red granular appearance; but when protru
congested, they constitute large tense tumid swellings of a d
colour and smooth surface, which readily bleed. When hæm
of large size are fully protruded, the integuments at the
of the anus become everted, and form a broad band girting the
the tumours externally.

External and internal piles often co-exist, the sphincter for
narrow band separating the two. But the two forms may me
each other, the difference being recognised by the character
covering, mucous membrane or skin, the line of junction being
on the surface of the tumours. Internal piles are confined
lower border of the rectum. They never occur, as has been
higher up the bowel, so that when they are entirely removed
very little liability to a recurrence of the disease.

Hæmorrhoids is a disease of middle and advanced age. They
occur before puberty, and but few persons in after-life al
escape them. All those circumstances which determine blood
rectum, or which impede its return from the pelvis, tend to
this disease. In many persons there is a natural predisposit
hæmorrhoids, and this may be hereditary. The complaint,
often occurs in members of the same family who inherit the
weakness of their parents. But a predisposition is more freq
acquired by sedentary habits, indulgences at table, and excite
the sexual organs, which explains the well-known circumstan
hæmorrhoids are more prevalent in the higher classes of societ
amongst the labouring population. The latter take plenty of e
live a good deal in the open air, and are little liable to cons
bowels. Hæmorrhoids, though common in both sexes, occu
frequently in males than females. Few women bear children
becoming in some degree affected by them; but the urinary and
disorders of the other sex, combined with freer habits of livi
still more fertile sources of piles.

The symptoms produced by hæmorrhoids vary a good deal
ferent subjects, and in different stages of the complaint. E
piles cause a feeling of heat and tingling at the anus. A
motion is followed by a burning sensation, and the excresce
comes slightly swollen and tender on pressure, so as to render
uneasy. This congested state of the pile may subside, or it m
to inflammation and considerable enlargement of the hæm
which then forms an oval tumour, red, tense, and extremely
The irritation produced by costive evacuations, or by frict
sitting and cleansing the part, sometimes gives rise to ulcer
the inner surface of the pile, causing a sore which extends
within the circle of the sphincter. This is liable to occur p
larly to those growths at the margin of the anus which hold a

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r severe, being a burning sensation lasting for some time after
on.

nal piles, when slight, may exist for years, causing little in-
ence besides slight bleeding after a costive motion, and occa-
a feeling of fulness, heat, and itching, just inside the anus.
small they protrude slightly with the mucous membrane in
on, returning afterwards within the sphincter. When of
size, they always protrude at stool and require to be re-
the patient usually pushing them up with his fingers. In
ate of the sphincters, and a loose and hypertrophied condition
mucous membrane from which they spring, piles come down,
hen the patient stands or walks about. When thus exposed to
ey appear very prominent, of a rounded form, and often of a
urple or violet hue, have a soft feel, and are evidently very
r, bleeding readily when handled. If free from congestion,
hibit a florid red colour, with a rough, granular surface. In
ience of the friction and pressure to which they are exposed,
ucous surface becomes abraded and furnishes a mucous dis-
tinged with blood, which soils the linen. They are often so
at the patient is obliged to lie down, sitting causing great
ess.

ns frequently suffer no inconvenience from piles until, irritated
stive motion, smart purgation, or the excitement of wine, they
congested and inflamed, and cause spasm of the sphincter
Patients then have what is termed an " attack of piles "—that
y, they suddenly experience a sensation of heat, weight, and
just within the rectum, followed by considerable pain at
d sometimes irritation about the bladder. Piles in this state
e to be strangulated and constricted by the external sphincter,
morrhoids of large size have been known to slough off, the
being relieved of the complaint by a sort of natural process,
uch pain and suffering. In general the extremities only of
two of the larger hæmorrhoids perish, and the patient, though
icing relief, is by no means cured of the complaint.

f the most common symptoms of internal hæmorrhoids, indeed,
m which the name of the complaint is derived, is hæmorrhage,
ccurs when the bowels are evacuated. The bleeding varies
in amount. Sometimes the motions are merely tinged with a
ps of blood ; in other instances the quantity lost is considerable,
ounces being voided at stool. The bleeding may be irregular,
g only after costive motions, or in certain states of health ; or
take place daily, going on even within the bowel, and pro-
the usual symptoms of derangement from continued losses of
The complexion becomes blanched, and the lips appear waxy.
ient loses flesh and strength, has a quick and small pulse,
from throbbings in the temples, palpitations and difficulty of
g on making any exertion, and at length the legs and feet

become œdematous. The character of the bleeding also varies; it is
sometimes venous, sometimes arterial. There are persons who are
liable to discharges of blood from the hæmorrhoidal veins either at
regular periods or when from good living or want of exercise the
habit is fuller than usual. In these cases from three to six ounces
of blood, or even more, pass away at stool, following the evacuation;
and the blood which is voided is of a dark colour and evidently
venous. Such discharges must not be rashly interfered with. I had
under my care a gentleman, seventy years of age, who had been subject
to hæmorrhoidal discharges for many years, usually in the spring and
autumn. After lasting a week or ten days they generally ceased, but
not always, and when faint and weak from their continuance, he was
in the habit of arresting them with cold water injections. The dis-
charges at length ceased, but in six months afterwards his urine
became albuminous, and a year later he died suddenly after an
attack of epistaxis. Periodical losses of this character relieve con-
gestion of the liver and kidneys, help to ward off attacks of gout
and prevent fits of apoplexy, so that in many persons they are rightly
regarded as safety-valves. Persons who suffer from internal piles
sometimes experience a pretty copious discharge of blood from the
rectum. The bleeding shortly ceases, and all uneasy symptoms sub-
side. This hæmorrhage is also venous. The escape of blood unloads
the congested parts and the patient gets relieved. But the bleeding
which most commonly occurs from internal piles is undoubtedly
arterial, taking place from arteries enlarged by disease. The vessels
on the spongy surface of the mucous membrane readily give way when
blood is determined to the part in defecation or when abraded by
the passage of hard fæces. An artery of some size, exposed by
ulceration, continues to pour out blood, weakening the patient, and
giving rise to the symptoms above described. Sometimes a small
artery on the prominent part of a protruded pile, may be observed
pumping out blood. That hæmorrhage of this character is good for
the health is quite a mistaken notion, and it is important that the
practitioner should distinguish the bleeding taking place as a con-
sequence of local disease from that which arises from a constitutional
plethora or congestion of intestinal organs.

When piles are small, and cause but little inconvenience, the treat-
ment is very simple. In all cases attention should be paid to the
habits of living. Persons with this complaint should take wine in
great moderation, if at all, and they are in most instances benefited
by abstaining entirely from stimulating drinks. Many individuals
never suffer from piles, except after taking a glass of spirits and
water, or a few glasses of wine. Such persons should become rigid
water-drinkers. Active exercise in the open air should be taken
daily, and the patient should avoid sitting too long at the desk,
because it is by prolonged sedentary occupation and neglect of the
rules of health that hæmorrhoid complaints are induced, which
explains why literary persons so often suffer from them. Chairs

with cane seats are to be recommended. The bowels must be care-
fully regulated, so as to avoid hard and costive motions, as well as
frequent actions. Irritating the rectum by repeated purging is more
hurtful even than constipation. On the other hand, when the liver
is congested, or its secretions are sluggish, and when the bowels are
costive, a mild cathartic, by clearing the intestines, especially the
large, unloads the congested vessels and relieves the piles. Lenitive
electuary, rendered more active when necessary by the addition of
the tartrate of potash, will probably answer the purpose. The
foreign mineral waters, the Püllna or the Friedrichshall, taken in
the morning, fasting, agree well with many patients, and ensure a
comfortable relief. When the intestines require fully unloading, a
draught containing rhubarb powder and the tartrate or sulphate of
potash answers without producing local irritation. Half a pint of
cold spring water thrown into the rectum in the morning after
breakfast has a very beneficial effect on the hæmorrhoids by con-
stringing the vessels and softening the motions before the usual
evacuation. The relief afforded by this treatment, combined with
care in the mode of living, is often remarkable. Ordinary venous
bleeding may be stopped in this way, using iced water, or some
astringent such as a solution of tannic acid or infusion of rhatany.
When the bleeding is of an arterial character, astringent injections
are not so successful, and operative treatment often becomes neces-
sary. When there is a slight slimy discharge from the surface of an
exposed internal pile, benefit may be derived from the application of
mild citrine ointment or the application of the solid sulphate of
copper to the part.

External piles, when large and troublesome, and internal, when of
such a size as to protrude at stool, and to be subject to inflammation,
ulceration, and frequent bleeding, can be removed only by operation.

PROLAPSUS OF THE RECTUM.—In describing the changes occurring
in piles, it was remarked that internal hæmorrhoids slip down and
project at the anus. The descent of these growths is often attended
with more or less eversion of the hypertrophied mucous membrane of
the lower part of the rectum. In relaxed states also of the sphincter
muscle and coats of the bowel, loose folds of mucous membrane are
liable to protrude and to require replacement. This protrusion and
exposure of the thickened mucous membrane with or without internal
hæmorrhoids has been erroneously described by writers as prolapsus
of the rectum. In the true prolapsus, however, there is a great deal
more than an eversion of the internal surface of the bowel. The gut
is inverted; there is a "falling down" and protrusion of the whole
of the coats—a change in many respects analogous to intussusception,
but differing from it in the circumstances that the involved intestine,
instead of being sheathed or invaginated, is uncovered and projects
externally.

The length of bowel protruded in prolapsus varies greatly, from an
inch to six inches or even more. The shape and appearance of the

swelling depend partly upon its size, and partly upon the condition of the external sphincter. When not of any great length, the protrusion forms a rounded swelling which overlaps the anus, at which part it is contracted into a sort of neck. In its centre there is a circular opening, communicating with the intestinal canal. An inversion of greater extent usually forms an elongated pyriform tumour, the free extremity of which is often tilted forwards or to one side, and the intestinal aperture assumes the form of a fissure receding from the surface of the tumour, owing to the traction exerted upon it by the meso-rectum. In a relaxed state of the sphincter the surface of the protrusion has the usual florid appearance of the mucous membrane; but in other cases it is of a violet or livid colour, and tumid from congestion, the return of blood being impeded by the contracted sphincter. The exposed mucous membrane is often thickened and granular, and sometimes ulcerated from friction against the thighs and clothes. A thin film of lymph may be occasionally observed coating its surface. On examining the section of a large prolapsed rectum from a child, I found the coats of the protruded bowel greatly enlarged; the areolar tissue was infiltrated with an albuminous deposit, the muscular tunic hypertrophied, and the mucous membrane much thickened and dense in structure, especially at the free extremity of the protrusion. These changes account for the difficulty in reducing the parts, and in retaining them afterwards, so often experienced in the treatment of this complaint in children, the bowel having become too large to be conveniently lodged in its natural position, and, like a foreign body, exciting the actions of expulsion. The atonic and relaxed state of the sphincter muscle in these cases is well shown by the facility with which one or two fingers can be passed through the anus even in children.

Prolapsus of the rectum is observed most frequently in children between the ages of two and four, but is liable to occur at a later period of life. In infancy it is produced by protracted diarrhœa; the frequent forcing at stool so weakening the coats and connexions of the rectum, and relaxing the sphincter, as at length to lead to inversion of the bowel. The straining efforts to pass water consequent upon stone in the bladder often give rise to prolapsus in early life. In adults the descent results chiefly from a weakened condition of the sphincter and levator ani muscles, and a general relaxation of the tissues of the part. The rectum being imperfectly supported by the perinæum, the eversion at stool gradually extends until an actual inversion takes place, and this may increase until it forms a protrusion of considerable size. Prolapsus in adults is more common in women than in men. In the former it results in a great measure from weakness in the parts consequent upon child-bearing.

The annoyance and inconvenience occasioned by a prolapsus of the rectum vary considerably under different circumstances. Thus the bowel may descend only in a very slight degree at stool, and disappear by a natural effort afterwards, or it may come down only occasionally,

mitting of being easily thrust back, and, when returned, will remain
its place until an attack of diarrhœa or the effort to pass a costive
)tion causes it to fall again. Prolapsus sometimes occurs after every
)tion, and even when the patient stands or moves about, forming a
ge red unsightly tumour exposed to friction, feeling sore, soiling
ə linen with a bloody discharge, and requiring to be pushed back
quently during the day. Or the gut may be constantly protruded,
ing fixed so as not to admit of replacement. There are cases on
:ord in which a prolapsed bowel has become strangulated and
flamed, and has even mortified and sloughed off, similar to what
metimes happens to an invaginated intestine.

Young persons generally outgrow this complaint by the period of
[berty; and common as is prolapsus in early life, it is rather rare in
own-up subjects. I have known, however, of persons, who have
d this disease in childhood, and lost it, becoming affected with a
turn of it in after-life from the effects of a diarrhœa. In adults
olapsus is commonly attended with a slimy discharge of mucus tinged
ith blood, and, in some instances, with troublesome bleeding. The
emorrhage does not occur from any particular spot, but as an
rudation from the congested mucous surface when the bowel is
rotruded at stool. As the cause producing the bleeding is con-
antly recurring, there is sometimes considerable difficulty in arrest-
g it, local applications having little effect so long as the bowel
ntinues to descend.

In children, irritability of the bowels and diarrhœa must be checked
d disordered secretions corrected by suitable remedies. Attention
ust be paid to diet, and when the powers are feeble benefit will be
rived from quinine or steel. In slight cases it will be sufficient to
rect the nurse, when the rectum comes down at stool, to place the
iild on its face across her lap, and to return the parts by taking a soft
ambric handkerchief or sponge wetted in cold water, in both hands,
nd by gentle but steady compression to push the protruded gut back
ito the pelvis. The relaxed state of the membrane may be corrected
y administering regularly every evening an astringent injection, such
s the decoction of oak bark with alum, the infusion of rhatany, or
he muriated tincture of iron diluted. The child should also be kept
t rest in bed, and be made to relieve its bowels in the recumbent
osture until the strong tendency to prolapsus has been corrected.
[he chief difficulty is to retain the parts after they have been reduced.
A piece of sponge or cotton wool, moistened in an astringent lotion,
nay be lodged at the anus and secured there by approximating the
nuttocks by means of a broad strip of adhesive plaster applied across
rom one side to the other, and further secured with a T bandage.
When the surface of the prolapsed bowel is ulcerated, it should be
ainted with a solution of nitrate of silver. In cases of stone, the
rrolapsus generally disappears after lithotomy.

Prolapsus in the adult requires surgical treatment to contract
he opening of the anus by escharotics or operation. In old and

unhealthy subjects the trouble may be remedied by a well-fitted rectum supporter.

IRRITABLE ULCER AND FISSURE.—The mucous membrane of the lower part of the rectum is arranged in longitudinal folds, which disappear in the expanded state of the bowel. These folds terminate below at the external sphincter. Just within this structure and between the folds, the mucous membrane is slightly dilated, variously in different subjects, but in many to such an extent as to form small sacs or pouches. Beside these folds, and in the spaces between them, there is a series of short projecting columnar processes, about three-eighths of an inch in length, separated by furrows or sinuses more or less deep, which are arranged around the lower part of the rectum. In the evacuation of the rectum, foreign bodies or little masses of hardened fæces are liable to be caught or detained in the pouches just described. It is in these little sinuses thus exposed to irritation, abrasion, and rent, that a superficial circumscribed ulcer is formed. On examining the ulcer without distending the rectum, the lateral edges only being presented to view, the breach of surface has the appearance of a *fissure*—the term commonly given, but improperly, to the sore, which though often originating in a rent is obviously more than a mere cleft or fissure in the mucous membrane of the bowel. Such an ulcer may occur in any part of the lower circumference of the rectum, but is usually found at the back part. It is quite superficial, and though sometimes circular is generally of an oval shape, its long axis being longitudinal and its lower extremity extending within the circle of the internal sphincter. On tactile examination the breach of surface and size of the sore can be readily distinguished. With the speculum, the ulcer being fully exposed is clearly seen not to be a mere fissure but a superficial sore. The surface is of a brighter red than the surrounding membrane, and has the usual indented appearance of an ulcer. A small pedunculated pile, or polypoid growth, attached to the opposite side of the bowel, is frequently found in these cases. The growth lodges in the ulcer, adding to the irritation and the difficulty of cure.

The amount of suffering produced by this superficial ulcer varies a good deal, but the sore is generally extremely sensitive and occasions severe distress. It is so situated that the fæces, in their passage outwards, rub over its surface, and the painful contact excites spasm of the sphincter muscle, causing a sharp burning pain, and often a forcing sensation, which lasts for two or three hours, the distress being usually greater after defecation than during the act; and in some instances, an interval, varying from five to ten minutes or more, elapses between the evacuation and occurrence of pain. The pain is sometimes so acute that patients resist the desire to pass motions, and allow the bowel to become costive in dread of the sufferings brought on by evacuating them. I have known persons to deprive themselves of food in order to avoid an action. In one case, the intensity of suffering led the patient to adopt the dangerous course of inhaling chloroform while

tting on the close stool, and he could not be persuaded to go to the
oset without this remedy.

The irritable ulcer occurs usually in middle life, and is more
quent in women than in men. It is met with as often in single
in married women. Though the symptoms are characteristic, the
re is often overlooked. On the attempt to separate the margins of
e anus, or to dilate the sphincter to get a view of the ulcer, or even
introduce the finger, spasm with an aggravation of pain is, in most
ses, immediately excited, and the orifice becomes strongly contracted
d forcibly drawn in. When this is the case, it is better to desist,
d to get an assistant to administer chloroform. Under its influence,
e sphincter yields completely, and the practitioner is able to ascer-
in the exact seat, character, and extent of the ulcer. In cases free
om spasm, a good view may be obtained by simply dilating the anus
ith the two forefingers, or by introducing a speculum.

The irritable ulcer seldom heals under the influence of local appli-
tions. The treatment necessary is an incision through its centre,
cluding the superficial fibres of the sphincter muscle, in order to
ace this muscle at rest, to enlarge the passage and displace the sore;
us removing those sources of irritation which prevent its healing.
n incision is not invariably required; but in all cases in which the
in is considerable, and in which there is much spasm of the
hincter, the attempt to procure the healing of the sore by local
plications so often protracts the patient's sufferings, and so con-
ntly ends in failure, that it is not desirable to make it. In cases
mplicated with a pedunculated pile or polypus, this growth must
o be excised. When the suffering is moderate, a cure may be
empted by giving a laxative to ensure soft evacuations, rest in the
umbent posture, and the application of mercurial ointment with
orphia, belladonna, or chloroform.

IRRITABLE SPHINCTER MUSCLE.—Persons occasionally suffer pain in
fecation, especially during solid motions, increasing afterwards, and
ting half an hour or an hour. It is described as a forcing sensation,
a feeling as if the bowel were unrelieved. The anus is strongly
ntracted and drawn in by the action of the sphincter. Any attempt
examine the part induces spasm; and the finger passed through it
tightly grasped by the muscle, as if girt by a cord. In cases of
l standing, the muscle becomes hypertrophied and forms a mass,
circling the finger like a thick unyielding ring. This irritability
d hypertrophy of the sphincter sometimes produces serious trouble
defecation, owing to the expulsive powers of the bowel being in-
fficient to overcome the impediment caused by this muscle to the
ssage of the fæces.

Irritability of the sphincter occurs commonly in hysterical females,
in nervous susceptible women, who are accustomed to watch and to
ensify every sensation. The treatment required is mild laxatives,
e local application of an ointment containing chloroform, opium, or
lladonna, and the occasional passage of a bougie coated with a

sedative ointment. The bougie gives great relief in those cases in which an irritable sphincter offers resistance to the passage of the fæces. In obstinate cases a partial or complete division of the sphincter may be necessary to remove the difficulty.

NERVOUS AFFECTIONS OF THE RECTUM.—The symptoms as well as the causes of these complaints are usually obscure, and the diagnosis is often perplexing. On analysing the symptoms, they appear to consist, in some instances, in an irritability, or a too frequent inclination to relieve the bowels; in others, in a morbid sensibility or undue tenderness of the part; and more rarely in an exaltation of sensibility independent of contact, constituting neuralgia.

1. *Irritable Rectum.*—In derangements of the alimentary canal, and of the organs connected with it, the fæces are often unhealthy and irritating to the mucous membrane; consequently when passed into the rectum they excite uneasiness, with an urgent desire to void them. Pressing and painful calls are also experienced when the bowel is ulcerated and in other ways diseased. In "the irritable rectum" there is an inclination, more or less urgent, to empty the bowel, usually at inconvenient times, although the mucous membrane, as well as the fæces, are healthy, and often when there is little or nothing to expel. Thus, a country rector experienced an urgent desire to relieve the rectum in church, just before and during the performance of divine service, notwithstanding an effort in the closet had just previously proved ineffectual. He was subject to it also when attending public meetings and whilst riding in a railway carriage. Persons living in the country and going daily to business by railway are sometimes annoyed by a desire to go to the closet just as the train is coming up, and during the journey to town, but it passes off as soon as they arrive at the counting-house and get engaged in business. The complaint is often connected with an anxious fidgety state of mind, against which patients may often successfully struggle. My patient, the rector, got relief from a gentle aperient on the Saturday, and a mild opiate suppository administered on Sunday morning.

2. *Morbid Sensibility of Rectum.*—Several cases have fallen under my notice in which uneasiness has been experienced at a particular spot in the rectum, being complained of, chiefly, during or after defecation. The fixity and sometimes severity of the pain, and its aggravation from pressure, have naturally led to the suspicion of the existence of some lesion in the mucous membrane, such as an ulcer: but on careful examination, no breach of surface has been discovered; nothing has been observed except in some instances slight elevations and increased redness and vascularity at the spot affected, and occasionally abrasion of the mucous membrane. The complaint consists chiefly in an exalted sensibility of the nerves of the part, but the alterations in appearance just described indicate that there is also some slight and superficial structural change. The remedies for the complaint are chiefly local. Sedatives, such as opium and belladonna, passed into the rectum give relief, but more permanent benefit may be derived from applications

culated to alter the character of the part, such as the sulphate of
per or a strong solution of the nitrate of silver applied through a
·culum. I have in several instances cured severe morbid sensibility
this part by two or three caustic applications.

3. *Neuralgia of the Rectum.*—The two forms of nervous affection
eady described would be included by some writers under the
eral term of *neuralgia*, the sensibility of the rectum being in a
asure perverted or augmented; but it will be remarked, that in the
it no actual pain is experienced—there is merely an irregular and
en causeless desire to evacuate the part; while in the second, the
easiness consequent upon the augmented sensibility is either pro-
ced or aggravated by friction and pressure. In true neuralgia of the
:tum, the pain is severe, but quite independent of contact. There is
tenderness. In the cases of neuralgia which have fallen under my
tice, the pain was not characterised by paroxysms, by a suddenness
attack and disappearance, or by any regular intermittence, nor was
e pain of an acute kind, but it was described as a continuous
during pain, or a constant gnawing sensation, sufficiently severe to
terfere seriously with the comforts and even the business of life.
e pain was in no degree mental, for the patients were not persons of
a anxious nervous temperament, and, unlike the two other forms of
rvous affection, occupation and amusement had little influence in
itigating their troubles. The remedies calculated to give relief are
ch as are useful in neuralgia elsewhere, as quinine, steel, arsenic,
omide of potassium, local sedatives, and hypodermic injections, and
ey are as uncertain in removing the affection of the rectum as in
ring neuralgia of other parts.

In some instances, it is impossible to refer nervous complaints of
e rectum to either of the forms just described, morbid sensibility
d neuralgia being so combined as to prevent any distinction being
rawn.

VILLOUS TUMOUR OF THE RECTUM.—A growth similar to the villous
mour which occurs in the bladder and on other mucous surfaces
metimes forms in the rectum. It was first described by Mr. Quain
der the name of a "peculiar bleeding tumour of the rectum;" but as
closely resembles the outgrowths found in the bladder called *villous*,
prefer the latter term. The tumour springs from the mucous mem-
ane generally by a broad base, is soft in structure, and composed of a
amber of projecting papillæ or villi. On minute examination it is
und to vary in structure according to the proportion of the fibrous or
scular elements entering into its composition. The villous tumour
innocent in character, and is not apt to return after complete
moval. Its chief peculiarity in the rectum as in the bladder is a
markable disposition to bleed. This growth is a rare disease, and
curs only in adults. When it projects at the anus, it exhibits
aracteristic projecting processes of a deep red colour.

The bleeding to which this growth gives rise and the slimy discharge
nder its removal very necessary. If the tumour be attached high

up, and a ligature can be applied round its base, this is desirable, as it would be difficult to arrest bleeding after excision.

POLYPUS OF THE RECTUM occurs in two forms—the *soft* or *follicular*, and the *hard* or *fibrous*. The soft polypus forms generally in early life. Its essential element is a considerable agglomeration of elongated follicles. There is a network of small vessels on its surface which is also furnished with papillæ. The polypus is attached to the mucous membrane of the rectum by a narrow peduncle which varies in length. The polypus is generally single, but several have sometimes been found. The follicular polypus usually makes its appearance external to the anus in children after a stool, and it resembles a small strawberry, being of a soft texture, granular on its surface, and of a red colour. It produces no suffering, but causes usually a slight bloody discharge, which occurring after every motion excites attention. In some instances the bleeding is sufficient to weaken the patient. The description of the complaint by the mother or nurse is apt to mislead the practitioner and to induce him to conclude that the case is common prolapsus. The growth can generally be detected by the finger passed into the bowel; and when the peduncle is long enough, the tumour is forced out at stool, and its nature can then be ascertained without difficulty. The follicular polypus occurs very rarely in the adult.

The treatment of polypus in children is very simple and always effectual. The tumour should be strangulated by a ligature secured around the pedicle and then returned within the bowel. This causes no pain, and the polypus comes away with the motions two or three days afterwards. Excision is not quite safe, as it is liable to be followed by bleeding.

The fibrous polypus is of a pear shape, with a peduncle more or less long and thick. It varies in firmness, seldom bleeds, but occasions a slight mucous discharge; and when the peduncle is long, or the tumour low down, it protrudes at the anus after stool, and requires replacement. When lodged within the bowel, it causes a sensation of unrelief, as if a foreign body or feculent lump required discharge. The polypoid growth sometimes becomes congested, and when protruded in this state its peduncle is liable to become girt by the sphincter, which causes great pain. The suffering is still greater when, as frequently happens, the polypoid growth is complicated with an ulcer within the circle of the sphincter. The polypus, coming in contact with the ulcer, irritates it, and prevents its healing.

The polypus must be removed by ligature or excision; and if an ulcer also exists, it must be divided at the same time.

FISTULA.—The loose areolar tissue around the lower part of the rectum is occasionally the seat of abscess, which bursts externally near the anus. But instead of the part healing afterwards like abscesses in other situations, the walls contract and become fistulous, and the patient is annoyed by a discharge from the opening. Such is the complaint termed *fistula in ano*. The abscess giving rise to fistula sometimes forms with all the characters and symptoms of acute phlegmon, suppuration taking place

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The chief symptoms referable to chronic ulceration of the rectum
are—a purulent discharge from the anus more or less copious; motions
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with blood; soreness in passing stools, and occasionally tenesmus.
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The character, position, and extent of chronic ulceration in the
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speculum with an open end made oblique and large. This instrument
is also very useful for the application of local remedies.

The treatment suitable to chronic ulceration greatly depends on the
nature and extent of the disease, and upon the constitutional con-
dition of the patient. In severe cases, I always keep the patient at
rest in the recumbent position. In extensive destruction of the
mucous surface with relaxed and copious discharges, especially when
the disease originates in dysentery, vegetable astringents, such as
simaruba, krameria, and bael, combined with the mineral acids and
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of its walls, forming the disease called *stricture*. The contraction may
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ments of defecation did not also interfere. Authors have described *blind internal fistula*, in which an opening into the bowel leads to a fistula without any external orifice. Such cases are rarely met with. The external opening sometimes closes for a short time, the spot being indicated by redness and induration; but sooner or later it reopens, and the discharge returns, or a fresh opening is made at some distance off. It may happen, however, that the original ulcerated opening in the rectum being large, the matter from the abscess in the areolar tissue outside finds its way so readily into the bowel that the abscess does not burrow towards the surface. The situation of the suppurating cavity may be ascertained externally by a sort of hollow or indistinct fluctuating feel. A bistoury plunged into this will render the fistula complete. A blind internal fistula is very liable to be overlooked. I have met with several instances in which this has happened. In one case, the discharge, which was abundant and kept the linen constantly soiled, was supposed to proceed from the vagina.

An anal fistula is at all times an annoying complaint. Even when the seat of the disease is free from all inflammation and tenderness, the patient is troubled with a discharge which stains the linen and keeps the part uncomfortably moist. The discharge is usually a thin purulent fluid; at other times it is thick, and in complete fistula tinged brown from admixture of feculent matter. The discharge is more or less copious in different cases, and varies also at different times. It occasionally becomes so thin and scanty that the patient supposes the fistula is about to close, when he is disappointed by fresh irritation being set up, and the complaint becoming as annoying as ever.

Anal fistula is a disease of middle life, and occurs more frequently in men than in women. It is occasionally met with in young children, but rarely forms in advanced life, owing partly to the laxity of the rectum and sphincter in old people rendering the mucous membrane less liable to irritation and injury, and partly to the relief obtained by discharges from the hæmorrhoidal veins when congested.

The treatment necessary during the formation of the abscess, which precedes the establishment of a fistula, is rest in the recumbent posture, fomentations or the hip bath, a poultice to the part, and mild laxatives. As soon as fluctuation can be felt, the prominent or central part should be punctured freely to prevent the matter burrowing in the loose areolar tissue, and thus to limit the extension of the sinuses. Fomentations and poultices must be continued until inflammation has subsided and the suppurating sac has become fistulous and indolent. An examination may then be made. This, as well as the cure of anal fistula by operation, is entirely surgical.

CHRONIC ULCERATION OF THE RECTUM.—The rectum is subject to ulceration in dysentery and other diseases, the mucous membrane being destroyed to a greater or less extent. Chronic ulcers of a tubercular character also occur in this part, but they are generally small in size. Several cases of ulceration in the rectum, the origin of which must be

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of the bowel. The submucous tissue is the chief seat of disease, and is condensed and converted into close-set fibrous tissue. The thickening of the coats of the bowel may be confined to part only of its circumference, or may be greater on one side than on the other, contracting the canal irregularly and forming a winding passage; or the induration, instead of being limited to a small portion of the bowel, may involve the greater part or the whole of the gut. The peritoneum investing the contracted bowel generally retains its healthy structure and appearance. Above the stricture the rectum is usually dilated and thickened. The enlargement results, not from a yielding of the intestine, but from a general hypertrophy of the walls of the bowel, and particularly of the muscular coat. The mucous membrane at this part is rarely healthy. It is red and tumid, or eroded and ulcerated, the diseased surface supplying during life a purulent discharge. There are often ulcerated apertures leading to fistulous passages which extend for some distance and open externally near the anus or in the buttock. The bowel below the stricture is generally more or less diseased, and frequently studded with small excrescences arising from partial hypertrophies or irregular growths of the surface and folds of the mucous membrane. These excrescences tend to narrow the canal below the stricture.

The seat of stricture in the rectum is at about an inch and a half to two inches from the anus, and easily within reach of the finger. In twenty-eight cases I found the stricture at this distance in twenty-one. In two it was nearer the anus, and in five at a greater distance. In three of the latter the stricture was at the point where the sigmoid flexure terminates in the rectum. In two instances I have met with double stricture.

The pathological changes causing stricture originate in chronic inflammation of the mucous and submucous areolar tissue of the rectum. It is seldom possible to fix on the exciting cause, but it is well known that the part is exposed to numerous sources of irritation. Women, in whom the disease is much more common than in men, have sometimes ascribed its origin to a difficult labour, by which no doubt the bowel may be injured, so as to set up chronic disease. In twenty cases of women with stricture of the rectum I ascertained that the disease commenced shortly after a labour, and in some instances was attributed to an injury at that time. Injuries such as a kick, and violent use of an enema tube, have also been known to give rise to stricture. Strictures sometimes originate in the contraction consequent upon the healing of ulcers or wounds in the bowel, more commonly indeed than is generally supposed. In extensive dysenteric and syphilitic ulceration of the lower bowel the passage is liable to become seriously contracted in this way. I have met with several cases of stricture of this kind.[1] The rectum may also be obstructed by an outgrowth of fat, or by an infiltration of fat in the coats of the bowel. This is a very rare form of stricture. There is a specimen of it in the

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Museum of St. Thomas's Hospital, and Mr. Worthington has related a case in the *Transactions of the Pathological Society* (vol. xv.). In the Museum of the London Hospital also there is a large fibrous and fatty tumour developed outside the rectum and contracting the passage.

Stricture of the rectum is a disease of middle life, being seldom met with in young persons except as a consequence of some injury. It is rare also in old people. The disease generally occurs between the ages of twenty and fifty.

The earliest symptom of stricture is, generally, habitual constipation with difficult defecation when the motions are solid. The difficulty being readily removed by a solvent purgative, the nature of the case is not usually suspected at this early period. As the contraction increases, the constipation is overcome with difficulty, and the patient acquires the habit of straining. The stools are observed to be small in calibre, and are often voided in small lumps. The mucous surface, irritated by the disturbance in the functions of the rectum, becomes inflamed and excoriated. This renders the actions of the bowels painful, a burning sensation lasting for a hour or more after stool. There is also a secretion of brown slimy mucus, which escapes with the motions and soils the linen. The gases evolved in the intestines not escaping readily, give rise to flatulent distension of the abdomen, especially in the course of the descending colon, and to disagreeable efforts for relief. The bowels often remain constipated for days together, and then a spontaneous mucous diarrhœa, excited by the fæcal collection or by a strong cathartic, softens the motions and enables the patient to void the accumulated mass, its passage being attended with pain. In other instances, the patient is teased with frequent fluid evacuations, and urgent desires to pass them. As the disease makes progress and ulceration ensues, the discharges become purulent and bloody, and the sufferings are much increased, the passage of motions being likened by the patient to a feeling as if boiling water was passing through the rectum. At this period, pain is often felt in the sacrum. The discharges are sometimes so copious that the stricture is overlooked, the case being mistaken for one of protracted diarrhœa. Ulceration often leads to abscesses and fistula, sinuses in the buttocks and labia being common complications of old-standing stricture of the rectum. The appetite and even the general health often remain good for a long time. The disease is very chronic; and so long as a passage for the motions can be obtained, the patient continues to follow his avocations, suffering more or less at different periods. The derangement of the digestive functions, the irritation kept up by the disease, and the exhausting discharges from the lower bowel in the course of time undermine the constitution and bring on hectic symptoms. The appetite fails, the body emaciates, profuse night-sweats ensue, and the stricture directly or indirectly becomes the cause of death. This is sometimes hastened by a lodgment of hardened fæces, or of some foreign body just above the stricture, so as to block up the bowel and occasion the symptoms of internal obstruction. Such an obstruction

is sometimes the cause of an examination of the rectum, and thus le
to the detection of a close stricture previously unsuspected.

In order to detect a stricture it is necessary to make a tac
examination. On exposing the anus small flattened excrescences
usually observed at the margin of the aperture. These cutane
growths resemble collapsed external piles, except that they are red
in colour, and are kept moist by the escape of a thin discharge f
the bowel. They originate in the irritation kept up by this discha
The finger, well greased, being passed carefully and gently into
rectum, will be arrested on reaching the stricture, so that the p
only can enter. If the contraction be somewhat recent and not v
close, the finger may be carried with a gentle boring motion thro
the stricture so as to examine its whole extent. If the practiti
encounters much resistance or gives much pain, he must not ven
to force the barrier, but must be content with ascertaining the
and degree of contraction. In strictures high up in the gut,
rectum below may be found quite healthy, but it is often dilated
baggy with weakened expulsive powers. In strictures low down
interior of the rectum is often abundantly studded with the
excrescences which I have described, which communicate to
finger the feeling of a number of rough irregular eminences,
or less hard, thickly lining the surface. This condition is inva
attended with a profuse discharge from the bowel of pus and
matter mixed with blood. A stricture high up in the rectum
beyond the reach of the finger is sometimes difficult of detect
In a suspected case the bowel must be explored by a flexible inst
ment. When the passage is free, a good-sized flexible gum ela
tube may always be passed into the colon. The point is apt
impinge on the sacrum, or to be caught in a fold of the bowel; but
some warm fluid, water or linseed-tea, be injected somewhat forcib
through the tube, a space is formed, admitting the easy transit of t
instrument. In stricture pain is felt when an instrument reaches t
point of contraction, and a flexible one is arrested or passed on wi
more or less difficulty. In examinations for stricture it must be bor
in mind that the rectum is liable to be compressed and obstructed b
disease of the neighbouring viscera—by an enlarged or retroflect
uterus, fibrous tumours of this organ, a distended ovary, an exces
sively hypertrophied prostate,—an hydatid tumour between th
bladder and rectum, or an outgrowth of fat, such as I have describe

The main object in the treatment of a stricture in the rectum is t
remove the chronic induration and to dilate the contracted part suffi-
ciently to admit a free passage for the fæces. The dilatation of the
stricture is to be effected by mechanical means—by the passage of
bougies, and sometimes by operation as well. The treatment, there-
fore, is chiefly surgical. An organic stricture fully established is
universally admitted to be most difficult of remedy, and several high
authorities, such as Dupuytren, Dr. Bushe, and Dr. Colles of Dublin,
doubt the possibility of the disease being cured. These writers have

ibtedly taken too unfavourable a view of the results of treat-
In addition to the dilatation, means must be adopted to relieve
ritability of the part, and to ensure the regular passage of soft
ations. An opiate suppository or injection may be lodged in
)wel at bedtime; and if the motions are costive, some confection
ina, castor oil, or Püllna water may be taken in the morning, in
just sufficient to obtain an action of the bowels without purging.
r oil is often of great service. In small doses it softens the
nt masses, and lubricates the passage without weakening the
it. Cod-liver oil is also an excellent remedy. It nourishes the
it and softens the motions, rendering aperients unnecessary.
liet should be nutritious and consist principally of animal food,
to afford a small amount of excrementitious matter. It is no
ess caution to advise patients to be careful to avoid swallowing
-stones. Accumulations in the bowel above the stricture may
evented by the occasional passage of an elastic tube through the
action and an injection of soap and water. We sometimes meet,
ially in hospital practice, with old, inveterate, and neglected
ures, in which the disease is too far advanced, and the mischief
reat, to admit of relief by dilatation. In such cases, when the
ings are severe, I have proposed the operation of lumbar-colotomy,
aave performed it in two cases.[1]

.ICER OF THE RECTUM.—The coats of the rectum are subject to can-
s degeneration in the three forms of scirrhous, encephaloid, and
d. The disease invades the coats to a greater or less extent,
icing contraction of the canal, and it is liable to increase until it
ws the passage to such an extent that only a probe can pass
igh it. Fungoid growths sometimes spring from the mucous
brane at the side of the rectum and project into the bowel.
sionally the bowel becomes blocked up and occluded by fungous
es. In other cases the changes which ensue have a contrary effect,
ieration and softening causing the coats to yield and increasing
alibre of the canal. A description of the progress of cancer of
ectum, and of the changes that occur in its advanced stage, is a
iption of the disorganization and invasion of all the tissues of
art, and of the organs in its immediate neighbourhood, in various
es in different cases. In some instances the carcinomatous bowel
nes wedged in the pelvis, agglutinated and fixed to the sur-
ling parts, forming one mass of disease. Frequently softening
ulceration cause fistulous communications with neighbouring
—with the vagina in the female, and with the bladder or urethra
e male; or the peritoneum may become perforated and an open-
nade into the abdominal cavity. When the passage is contracted,
intestine above becomes dilated and hypertrophied as in simple
ure. Carcinoma may attack any part of the bowel, but it gene-
affects the lower portion within three inches from the anus. It
ible to occur also, though less frequently, at the point where the

[1] *Vide* London Hospital Reports, vol. iii.

sigmoid flexure terminates in the rectum. The disease is sometimes limited to the rectum and adjoining parts, though the lymphatic glands in the pelvis and lumbar region often become affected, the liver being invaded by tubercles, and the peritoneum also studded with scirrhous deposits.

Cancer of the rectum generally commences insidiously. Its early symptoms are so similar to those of simple stricture, that the nature of the disease cannot be determined, or may not be suspected, until a considerable change has taken place in the condition of the bowel. The patient is troubled with flatulency, has difficulty in passing his motions, and strains in the effort to void them; and as the disease makes progress, he experiences pains about the sacrum, which gradually increase in severity and dart down the limbs. By this time probably some alarm is excited, and an examination may be called for. The practitioner on introducing his finger into the rectum may easily detect a contraction more or less rigid; and should he feel any irregular nodules about the stricture, any hard solid tumour, or encounter a resistance like cartilage, or meet with softish tubercles which leave a bloody mark on the finger, then he would be able to decide on its being carcinomatous. At a later period no difficulty could be experienced. There is a hard mass of disease in which it may be difficult to discover the orifice of the passage, and sometimes round fungoid growths which bleed readily when touched. The disease may extend as low as the anus. An irregular red-looking growth sometimes protrudes externally, blocking up the passage or displacing the anus. The stools become relaxed and frequent and contain blood, and in passing cause a scalding pain and give rise to severe suffering. There is often a thin offensive discharge, and as the disease invades the sphincter incontinency ensues. The loss of retentive power is often a great trouble in cancer of the rectum. This arises not only from the disease invading the anus and destroying the sphincter muscle, but occurs also when cancer is developed higher up in the bowel, the lower part being free. This may be explained by the carcinomatous disease pressing or destroying the nerves supplying the sphincter and so paralysing it. The sufferings also increase. Severe shooting pains are referred to the groins, back, or upper part of the sacrum, and sometimes extend down the thighs and legs. The constitution suffers in due course. The patient acquires the blanched sallow look, anxious countenance, and emaciated appearance commonly observed in persons suffering from malignant disease. If complete obstruction does not accelerate a fatal termination, other troubles may arise. In consequence of a communication becoming established between the rectum and urethra or bladder in males, flatus and liquid fæces escape from the urinary passage, and in females motions are discharged from the vagina. The passage of part of the intestinal contents by these unnatural channels greatly increases the misery of the patient's condition, rendering him an object of disgust to himself and offensive to those

about him. An ulcerated opening into the peritoneum, allowing the escape of feculent matter into the abdomen, may excite peritonitis and thus bring the case to a fatal termination; or the powers of life gradually giving way, the patient becomes hectic and exhausted, worn out by this painful and distressing malady. There is great variety, however, in the degree of suffering, and even of constitutional derangement, attending the disease. Whilst in some cases the sufferings are excruciating, in others they are comparatively slight. In my experience patients suffer less from the disease when developed high up in the rectum than when formed near the anus.

Cancer of the rectum occurs generally in middle life. The earliest age at which I have met with it is twenty, the patient being a young man in the London Hospital. It is commonly believed that this disease attacks women more frequently than men. This does not accord with my experience of cases seen in hospital and private practice. Of seventy-three cases of which I have preserved notes, fifty-seven were males and sixteen females.

All that can be obtained from remedies is palliation of the symptoms, ease from pain, and support under the wearing effects of this terrible disease. The patient should remain at rest, chiefly in the recumbent posture, and take a nourishing but not stimulating diet. The general health may be supported by tonics. The bowels must be kept open and the motions rendered soft by Püllna water, or small doses of castor oil. If the stricture be close, injections may be necessary through a long tube to break up the feculent masses. The greatest care is necessary in the passage of the tube, as if force be used the carcinomatous mass may yield and the tube be driven into the abdomen. Bleeding may be checked by injections of sulphate of copper or tannic acid. Pain can be alleviated by opiate and belladonna injections, or by small doses of morphia taken night and morning, their strength being gradually increased as the effects of the remedy diminish. Subcutaneous injections of morphia also are effectual in giving relief. So great were the sufferings in a recent case that after a time as much as $3\frac{1}{2}$ grs. were thus injected twice a day.

In cancerous disease of the rectum attended with great suffering from incontinency and constant scalding discharges, I have advocated and performed in several cases colotomy in the left loin. By diverting the passage of the fæces, the local distress can be in a great measure prevented, and I have reason to believe that the progress of the disease also may be retarded by the removal of a source of almost continual irritation. I have established an anus in the left loin in several cases of cancer in which no obstruction existed, in order to mitigate the symptoms, with a satisfactory result in prolonging life and preventing suffering.[1]

EPITHELIAL CANCER OF THE ANUS AND RECTUM.—The anus, like other parts, where a junction takes place between the skin and mucous membrane, is liable to epithelioma. The affection is comparatively

[1] *Vide* London Hospital Reports, vols. ii. and iv.

rare, and has seldom been noticed by writers. It is easily recognised by the ordinary characters of the sore. In the few cases which have fallen under my notice, the disease extended into the rectum, but there was no reason to doubt that its original seat was the anus. The only treatment applicable to this affection is caustics or excision. I prefer the latter, as more sure and thorough. Though more common at the anus, epithelioma may occur in any part of the mucous membrane of the rectum. When occurring up the bowel, the disease is apt to produce slight bleeding, but it is much less serious than scirrhous and medullary cancer. The latter produce sooner or later some contraction or obstruction in the passage, and show a tendency to involve the parts around. In epithelial cancer I have never noticed any impediment in defecation, and have invariably found the passage free and unobstructed. Neither do patients complain of the distressing pain, referred usually to the sacrum, which persons affected with scirrhus of the rectum so commonly experience, nor suffer painful tenesmus and defecation, which add so much to their distress in this form of the disease. There is also an absence of the cancerous cachexia, of the emaciation and pale and anxious countenance so frequently remarked in malignant disease. Epithelial cancer in the rectum may go on for years, but the patient becomes exhausted at last from repeated small bleedings. The hæmorrhage is best restrained by injections of solutions of sulphate of copper, chloride of zinc or tannin.

ATONY OF THE RECTUM.—In paraplegia the forces which expel the fæces and the retentive functions of the sphincter are both destroyed; consequently, the motions, if sufficiently liquid, on reaching the lower bowel escape involuntarily. I have not met with any well-marked case of paralysis of the rectum independently of palsy of the lower half of the body; but several instances of loss of tonicity or defective muscular power in the lower bowel, rendering it incapable of properly extruding its contents, have come under my notice. An atonic condition of the rectum may be produced by the too free and frequent use of enemata, the quantity thrown up being so large as to dilate the bowel and impair the power of its muscular coat. This condition is apt to give rise to fæcal accumulations. Cases of this kind are not very uncommon, yet they are liable to be overlooked by practitioners. It appears that the rectum becomes gradually dilated and blocked up by a collection of hard dry fæces which the patient has not the power to expel. Some indurated lumps from the sacs of the colon, on reaching the rectum, perhaps coalesce so as to form a large mass; or a quantity accumulated in the colon on descending into the lower bowel becomes impacted there. In several instances a plum-stone has been found in the centre of the mass. Such a collection gives rise to considerable distress and alarm, producing constipation, a sensation of weight and fulness in the rectum, tenesmus and forcing pains. In cases of some duration when the hardened fæces do not quite obstruct the passage, they excite irritation and a mucous discharge which, mixing with recent feculent matter passing over the lump, causes the case

to be mistaken for diarrhœa. Injections have no effect in softening the indurated mass. They act only on the surface and return immediately, there being no room for their lodgement in the bowel. On digital examination the bowel is found to be distended and blocked up with a large lump which feels almost as hard as a stone. In such cases, the only mode of giving relief is by surgical interference. The mass requires to be broken up and scooped out. Sir James Simpson has described this affection under the head of " ball-valve obstruction of the rectum by scybalous masses."[1] Some years ago I saw a lady who for eighteen months had been unable to relieve her bowels without aperients and without passing her finger into the rectum. On examination I detected a hard elongated mass which was forced down in the effort of defecation and obstructed the anus until the finger pushed it back. I broke up this mass, and after the bowels had been relieved by injections the difficulty was entirely removed.

ANAL TUMOURS AND EXCRESCENCES.—Besides the flaps and folds of integument consequent on external piles, other growths are developed in the immediate vicinity of the anus. These tumours of a fibrous texture sometimes form in the subcutaneous areolar tissue, and as they increase become pedunculated. They seldom exceed the size of a chestnut, though I have known one to weigh half a pound. They have a firm feel, and their surface is in general irregularly lobulated. These growths may be easily and safely removed by excision.

Warts are not unfrequently developed around the anus, and they sometimes grow so abundantly as to constitute a considerable cauliflower-looking excrescence. They then form projecting processes of various sizes densely grouped together, many being of large size, with their summits isolated, expanded, and elevated on narrow peduncles more or less flattened. I have seen a mass forming a tumour as large as the closed fist, separating the nates, and almost blocking up the passage for the fæces. When abundant, they give rise to a thin offensive discharge. They originate in the irritation consequent on want of cleanliness, and occur generally in young adults of both sexes. I once saw a large crop of these growths in a child only four years of age. In some persons there is so strong a disposition to the formation of warts, that without great attention it is difficult to prevent their formation. If few in number and small in size, they may be destroyed with strong nitric acid. They usually require however to be removed by excision, which is the quickest and most effectual mode of treatment. Great cleanliness and the application of astringent lotions will be necessary to prevent their reproduction afterwards.

PRURIGO ANI.—Itching at the anus is a common symptom in several disorders of the lower bowel, but it may also occur as a distinct affection, as independently of any other disease of the part, being due to a peculiar hyperæsthesia of the skin. Prurigo ani is caused by worms in the lower part of the rectum, and by congestion of the

[1] Edinburgh Monthly Journal of Medical Science, April 1849.

hæmorrhoidal veins. In women it is consequent on affections of the womb. Patients suffer most after taking stimulating drinks, and during warm weather and when heated in bed. The itching is extremely teasing and annoying, especially at night, when it sometimes keeps the patient awake for hours. Rubbing the part to arrest the irritation only aggravates the mischief afterwards, yet few persons have sufficient self-control to prevent their seeking temporary relief by friction, and some, though capable of restraining themselves whilst awake, fret the part unconsciously during sleep. The friction thus resorted to excoriates the skin at the margin of the anus, so that in chronic cases the skin becomes dry, harsh, and leathery, cracks from slight causes, and ulcers and fissures are produced, which are but little disposed to heal. In most instances this complaint, after proving troublesome for an hour or two at night, and in the day after stimulants, ceases, and the patient has long intervals of rest and ease. But in the worst forms of the malady, the torment is most distressing. It lasts throughout the night, so that the patients get little but broken sleep, and after a time the general health suffers seriously, and life is rendered truly miserable. In some of the cases which have fallen under my notice, I could discover no local cause whatever to account for the prurigo. It seemed to be purely an affection of the nerves of the part. The patients are generally healthy. One gentleman who had been subject to it for years, found that it was connected with his state of mind. When much engaged and prosperous in business, he suffered little from it. He was sometimes free for a whole month, and then became troubled for many nights in succession. In cases of this kind the complain , after proving troublesome for years, has been observed to subside as age advances.

In prurigo ani the habits of living should be regulated. The patient should sleep on a mattress, and be as lightly covered as is consistent with comfort, cold bathing or sponging should be daily resorted to, and sufficient exercise taken in the open air. Stimulants and hot condiments must be strictly avoided. The actions of the bowels are to be regulated if necessary by medicine, and after each evacuation the parts should be cleansed with soap and water. Every effort should be made to avoid friction, and the patient should be assured that if he yields to his inclinations, his complaint will be rendered worse and more difficult of cure. In all cases, the condition producing this troublesome symptom must be the chief object of attention, such as worms, congestion, &c., but there are certain remedies which are specially adapted to relieve the irritation. The itching attendant on piles may be arrested by smearing the anus with some mercurial ointment, as the dilute citrine, or one containing the grey oxide of mercury, or by lodging in the parts a piece of cotton wool soaked in a lotion of oxide of zinc. Lotions of carbonate of bismuth and glycerine, of borax and morphia, or of carbolic acid, are often efficacious in this complaint. The application to the anus of strong solution of nitrate of silver (gr. xx—$\bar{3}$ j) with a camel's-hair brush

e daily often gives relief, especially in cases where the skin is
le rough and sore by rubbing.   In some cases great benefit has
n derived from chloroform ointment.   It produces a smarting sen-
on when first applied, but this is soon followed by ease.   In persons
weak constitution benefit has resulted from full doses of quinine,
. in certain cases liquor arsenicalis with steel has helped to relieve
irritation.   I have sometimes found it necessary in severe cases to
er suppositories of morphia at bed-time.   The complaint is often
y obstinate, and much perseverance is required on the part of the
ctitioner, and also of the patient, to effect a cure. .

INTESTINAL WORMS.

By W. H. Ransom, M.D., F.R.S.

Introductory Remarks.—No definition of the disease,
stands at the head of each article in this System of Medi
requisite or appropriate in treating, from the point of view
practical physician, of the parasitic worms which inhabit the
alimentary canal. But it may be desirable briefly to indic
general scope or plan of this article, as well as the limits
which it will be restrained.

In most diseases, as for instance in the exanthemata,
summary of the more constant phenomena may serve at on
definition and means of diagnosis; but, as the external agents or
causes of those phenomena escape our search, the etiology
diseases is little more than an investigation of the conditions
able to their occurrence, with speculations upon the nature
exciting cause; while the pathology is limited to a considera
the relations existing among the phenomena observed during
after death, and between these and the favouring conditions.

But in the medical study of parasites the whole question
changes from a condition of health" is viewed from quite anothe
point. Here we can begin with the exciting cause, which
isolate, compare, experiment upon, and learn the natural his
before we study its effects. The extension of knowledg
possibly hereafter enable us so to approach the study of ch
scarlet fever.

In this article the order thus indicated will be followed; the
and zoological position of the worms found in human intestine
first stated, the more important species will be described and th
histories traced, with only so much of detail as may be requi
the purposes of the medical practitioner. Afterwards the cha
function or structure which they produce, the conditions which
their occurrence, the mutual relations of the observed phe
the methods of detecting, expelling, and avoiding these pests,
treated of.

Those parasitic animals belonging to the Gregarinida and In
as well as the accidental or occasional but not truly
inhabitants of our intestines, such as insect larvæ, will be e
from consideration here on account of their at present eom

ificance clinically. The *Trichina spiralis* will also be passed
because, although it attains its state of sexual maturity in human
nes, its importance to the physician depends upon the habit
its larvæ have of perforating the tissues and becoming encysted in
iscles. Moreover the very great importance which has recently
ed to this worm justifies the devotion to it of a separate article.
s difficult, if not impossible, adequately to appreciate the relation
estinal worms to their bearers without including in the investi-
the lower animals. To do so here would, however, be foreign
design of this work, and the reader who seeks for fuller
iation on this subject will do well to consult the works of
enmeister, Von Siebold, Davaine, Cobbold, and especially of
art. I may however draw attention to two prominent results
comparative study of Entozoa. They are so widely diffused
carcely any species of animal is known which is not, at least
imes, infested by them; and notwithstanding the fact that they
nd do, often injuriously and even fatally influence the animals
infest, yet in the majority of cases the observer is struck with
parently trivial inconveniences they produce.

TORY.—The intestinal worms, or some of them, have been known
very early times. Hippocrates mentions the tape-worm, and
otle described in addition the round-worm and the seat-worm.
ig the classical and middle ages the doctrine of spontaneous
ation held general sway, and was thought to afford a satisfactory
nation of the then known facts as to the occurrence of Entozoa.
ugh Swammerdam [1] and Redi [2] shook the foundations of this
ne in its application to insects and their larvæ, they did not
re to apply their views to the Entozoa. The first great step
ds sounder views was made by Pallas,[3] who taught that
oa, like other animals, sprang from similar parents, and were
gated by means of eggs which were transmitted from one host
ther. But in the absence of direct evidence these opinions were
ime borne down by the authority especially of Rudolphi [4] and
ier,[5] who reverted to the doctrine of spontaneous generation.
however, the progress of biological science, aided by improved
of research, and directed into new channels, broke down this
ne at once and for all time, at least in its application to intestinal
s; and the researches of Mehlis (1831),[6] Von Siebold (1835),[7]
Eschricht (1837)[8] confirmed the main proposition of Pallas,
ustified the conclusion of Eschricht, that Entozoa during their

[1] Bibel der Natur. Aus dem Holl. übersetzt. 1752.
[2] Esperienca intorne agl' Insetti. 1712.
[3] Neue Nord. Beiträge. 1781.
[4] Entozoor, hist. Natur, vol. i. 1808.
[5] Ueber lebende Würmer im lebenden Menschen. 1819.
[6] Oken's Isis. 1831.
[7] Archiv für Naturgeschichte. 1835.
[8] Nova Acta Academ. C. L., vol. xix. 1837.

reproduction generally undergo a metamorphosis and a migration
Then followed the brilliant discovery of alternation of generations b
Steenstrup (1842),[1] the researches of Von Siebold (1848),[2] and Va
Beneden (1850),[3] and the true life history of the *Trematoda* a
Cestoda was understood. It remained to furnish direct proofs of th
correctness of the new views, and these were given by Kückenmeist
(1852),[4] who fed carnivora on flesh containing *Cysticerci* and produc
tape-worms, and by feeding herbivora with ova of Tæniæ produc
Cysticerci. Many other zealous and able investigators in this countr
as well as in France and Germany, have confirmed his results, an
otherwise extended our knowledge of the intestinal worms. Promi
nent among these stand the names of Haubner, Leuckart,[5] Dujardi
Davaine,[7] and Cobbold.

The opinions of medical men as to the clinical importance
intestinal worms have varied with the changes of biological theo
usually lagging somewhat behind, but depending mainly upon it.
long as the doctrine of spontaneous generation in any of its form
was believed to account for the presence of Entozoa a mysterio
dread of their power for evil prevailed, and evidenced itself by t
multitude of grave diseases attributed to them. Indeed few mala
afflict humanity which were not sometimes attributed to intesti
worms, even by prominent men in their day.

This was due not alone to the common tendency to magnify
unknown, but also to the uncertainties of diagnosis, the absenc
a pathological anatomy, and the frequency with which worms w
observed to pass away in the course of serious diseases, the subsequ
recovery from which being imputed to their escape.

In the latter half of the eighteenth century an extreme reaction too
place among those who gave themselves specially to the study
Entozoa, so that it was maintained that they were beneficial to th
hosts, or at most only very rarely and accidentally injurious.

The physicians as a rule, however, still clung to the older view
and in doubtful cases found a ready and satisfactory explanation
the symptoms in the assumption of an irritation by imaginary worm
Even Rudolphi and Bremser, while opposed to the prevalent medic
opinion, sought to explain the actual symptoms which attended th
presence of worms in the intestines by the hypothesis of a pre-existin
diathetic state (Helminthiasis), which they believed to be a necessar
condition of the spontaneous development of worms. Only in the prese
generation have sound views on this subject prevailed, and only sin
the discoveries of Kückenmeister and his followers has a satisfactor
knowledge of the life history of human intestinal worms enabled th

[1] Ueber den Generationswechsel. 1842.
[2] Jahresbericht im Archiv für Naturgeschichte. 1848.
[3] Les Vers Cestoides. 1850.
[4] Prager Vierteljahrschrift. 1852.
[5] Die menschlichen Parasiten, &c. 1852-68.
[6] Histoire Naturelle des Helminthes. 1845.
[7] Traité des Entozoaires. 1860.

hysician to appreciate their true importance in medicine, to ascertain
eir presence with certainty, and in most instances to point out how
ey may be avoided.

Out of at least thirty-one Entozoa which are at present known to
habit our bodies, thirteen infest the alimentary canal. Of these
ven belong to the order *Cestoda :*—

1. *Tænia solium*, Linnæus.
2. *Tænia medio-canellata*, Kückenmeister.
3. *Tænia nana*, Von Siebold.
4. *Tænia flavo-punctata*, Weinland.

5. *Tænia elliptica*, Batsch.
6. *Bothriocephalus latus*, Bremser.
7. *Bothriocephalus cordatus*, Leuckart.

nd six to the order *Nematoda :*—

1. *Ascaris lumbricoides*, Linnæus.
2. *Ascaris mystax*, Rudolphi.
10. *Oxyuris, vermicularis*, Bremser.

11. *Dochmius duodenalis*, Leuckart.
12. *Trichocephalus dispar*, Rudolphi.
13. *Trichina spiralis*, Owen.[1]

ORDER *CESTODA.*

Parenchymatous worms, without mouth or alimentary canal, with
a so-called water-vascular system. They develop by budding
from a pear-shaped larval form (scolex) to a long, jointed, tape-
shaped colony of individuals (strobila). In their reproduction they
suffer an alternation of generations. The individual members of
the colony (proglottides), or sexually ripe animals, increase in size
and complexity of structure, although otherwise resembling each
other, the further they are removed from the head, near to which
a continuous formation of new joints takes place by budding. The
head, which is the same in the adult as in the larval form, is furnished
with two or four suckers, and commonly also with a coronet of
booklets, which serves for attachment. They infest in their adult
state the alimentary canal of vertebrate animals only. The ovum
yields a globular embryo furnished with three pairs of hooklets, and
develops into the Scolex (*Cysticercus*) in the tissues or in parenchy-
matous organs, usually of food animals, and is thence passively trans
ferred with the food into the intestine of its definitive bearer, where
it assumes the adult form.

TÆNIA SOLIUM (Linnæus)

Was at one time believed to be " the common tape-worm of man," but
it is now known that at least one other species is included in that
expression.

Description.—The adult worm (Strobila, Fig. 1) commonly attains
a length of from 7 to 10 feet,[2] but is often much longer. The number
of joints increases with the length ; a worm measuring 7ft. 6in., counted

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[1] See article *Trichina spiralis*.
[2] This is Leuckart's measurement, but there is a wide divergence among authorities on
his point. Davaine makes the common length from 20 to 26 feet.

by Leuckart, had 749 joints. The head (Fig 2) has a som
globular form, measures about $\frac{1}{45}$ in. to $\frac{1}{35}$ in., is marked anteriorl
moderately prominent rostellum, bearing a crown of about twen
hooks, and by four projecting suckers.

The threadlike neck is nearly an inch in length, and to the
eye is not distinctly jointed; it passes gradually into a jointed
tinually widening band of a whitish colour, of which the
segments are so much shorter than broad that one-half of the

FIG. 2.—Head of *T. solium.*
(Davaine.)
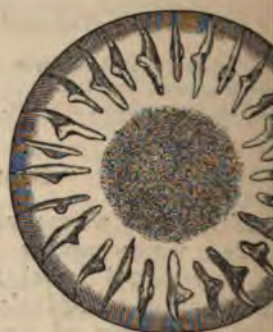
FIG. 3.—Coronet of hooks, magn
(Leuckart.)

FIG. 1.—*Tænia solium,*
natural size. (Davaine.)

FIG. 4.—Separate hooks, more highly magnified.
(Leuckart.)

are found in the anterior ninth of the chain. Slowly the j
increase in length more than in breadth, so that they assume a s
form about the end of the anterior third. Mature joints, *Proglo*
or *Cucurbitina* (Fig. 5), measure about $\frac{1}{2}$ in. in length and $\frac{1}{4}$ in. in bre
being now longer than broad. They are flat and thin, with a
rangular outline, are furnished with a longitudinally placed tu
uterus, having seven to ten branches on each side, within whic
seen developing ova. Male and female organs of generatio
present in the same joint, and open by a common aperture nea

ntre of one or other border, now right, now left. The sexual organs
e already distinctly visible in the joints at one-ninth of
ie whole length from the head, the ova are impregnated
bout another ninth lower down the chain, and soon after-
ards the eggs enter the uterus.

The water-vascular system consists of a single longitu-
inal canal at each border, and one transverse, near the
osterior edge; it is continuous from one segment to
nother throughout the chain. The cystic worm known
is *Cysticercus cellulosæ* is the larval form, or *Scolex* (Figs.
6, 7); it is commonly found in the flesh of pigs, but
occasionally also in other animals, and even in man: the
adult colony has only been found in man. The eggs
(Fig. 8) are globular in form, measure when free about
¹⁄₃₀ in., have a thick firm shell of a brownish colour, radi-
ally and concentrically striated, and when taken from the
uterus often an outer capsule with a more oval outline
(Fig. 8 *a*). The contained embryo is globular, and fur-
nished with three pairs of hooklets. A moderate-sized
tape-worm has been calculated to contain about 5,000,000
of ripe ova.

Fig. 5. — Ripe joints of *T. so-lium*, magni-fied. (Leuck-art.)

Life History.—The normal habitat of *T. solium* is the small

intestine of man: Kücken-
meister has seen it while
yet alive firmly attached by
suckers and coronets to the
mucous membrane. For-
merly it was believed that
it was always solitary, and
this error perhaps explains

Fig. 6. — *Cysticercus cellulosæ*, natural size and position. (Leuckart.)
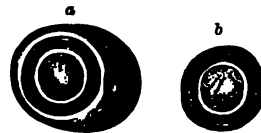
Fig. 8.—Ripe ova of *T. solium a* with outer capsule as seen in uterus: *b* free, as found in fæces. (Leuckart.)

the statements made by the older authorities of the occurrence of
worms of enormous length. It is now known that although commonly
one, two, or three are found together, yet various numbers, up to forty
at least, may be present.

From the lowest end of the band—which hangs
a variable distance down the intestines, and may
reach the colon—ripe joints spontaneously sepa-
rate and escape with the fæces, either singly or
united into short lengths. Frequently, also, ripe
ova escape by rupture from the joints into the
intestine and mingle with its contents. The
free joints in moist and warm situations move
about for a time, and by this and other accidental
agencies the ova are widely disseminated; doubt-
less the vast majority fail to find suitable con-
ditions for their development, and therefore die;

Fig. 7.—*Cysticercus cellu-losæ*, magnified. Head and neck protruded. (Leuckart.)

but a small proportion of joints or ova are taken
with the food into the stomach of a pig, or much more rarely

that of a man; where, after digestion and rupture of the shell, the embryo (*pro-Scolex*) escapes, and by diligent use of its armature perforates the tissues of its involuntary host, and ultimately settles down in some, to it, suitable locality, generally the cellular tissue of the muscles, but sometimes the liver or the brain. The embryo there remains quiet, in some organs is encysted, undergoes a metamorphosis, and becomes the well-known *Cysticercus cellulosæ* of measly pork (Figs. 6, 7). As usually found, it has the head and neck inverted, and its characters are difficult to observe, but when everted is seen to have a head and neck like that of *T. solium*, with a vesicular caudal appendage. This metamorphosis requires about two months and a half for its completion; afterwards the *Cysticerci* remain without further change, but capable of further development, if the proper conditions are supplied, for a period not yet certainly known, but which has been estimated at from three to six years.

When the flesh of pigs so infested is eaten raw or imperfectly cooked, the *Cysticercus* is partly digested in the stomach, so as to lose its vesicular annex; it then passes into the small intestine, and attaching itself, becomes developed in about three to three and a half months into the adult form already described, which may continue to infest its bearer for ten or even, it is said, thirty-five years. It would take too much space here to recount the evidence upon which this summary statement rests; but it may be said in brief that Kückenmeister, Leuckart, and others have, notwithstanding some opposing statements, placed it beyond reasonable doubt by a carefully devised and executed series of experiments, in which pigs have been infected with *Cysticercus cellulosæ* by eating ripe joints of *Tænia solium*, and men have been infected with tape-worm by eating measly pork.

This biography of *T. solium* illustrates that of other parasites of the same group, and the study of each has thrown light upon the others; for this reason, and to show the relation between the food of animals and their parasites, the following short list may be permitted a place here:—

Cysticercus fasciolaria in the mouse is the larval form of *Tænia crassicolles* in the cat.

Cysticercus pisiformis in the rabbit is the larval form of *Tænia serrata* in the dog.

Cysticercus tenuicollis in sheep, oxen, &c., is the larval form of *Tænia marginata* in the dog.

Cœnurus cerebralis in sheep is the larval form of *Tænia cœnurus* in the dog.

Cysticercus tæniæ medio-canellatæ in the ox is the larval form of *Tænia medio-canellata* in man.

Symptoms.—There can be no question that a large proportion of persons infested with this tape-worm are unconscious of any departure from the state of perfect health, but there is as little doubt that in some instances functional derangements occur which are referable

he local irritation it produces. In a much smaller number of cases, and under exceptional conditions, even structural changes are produced by it.

The functional derangements belong to two groups. (a) Those excited in the part irritated, and its immediate neighbourhood. Such are, various uncomfortable sensations in the abdomen, pains resembling colic, sometimes felt when the stomach is empty, at others after certain articles of food, variable appetite, now excessive, now failing entirely, slight diarrhœa, or constipation, &c. (b) Those of reflex origin. These are itching of the nose or anus, headache, giddiness, ocular spectra, tinnitus aurium, palpitation, cardialgia, increased flow of saliva, nausea, lassitude, pains in the limbs, and an uncertain flow of spirits. In women, disordered menstruation, spasmodic and convulsive movements, hysterical fits, and even epileptic and maniacal attacks, have been said to be due to their irritation. In long-continued cases, Kückenmeister thinks wasting has been produced. This somewhat grave list of symptoms contains little or nothing that is characteristic of the nature of the irritative cause, and must be received with some caution, on two grounds: one, that patients not unfrequently exaggerate their sensations when they either have had, or have suspected themselves to have had, worms of any kind; and the other, that the symptoms enumerated have in great part been collected and handed down to us from earlier times, when medical men, not yet familiar with the results of comparative helminthology, shared, to some extent, the common mysterious dread of Entozoa, and too hastily attributed the observed phenomena to the influence of worms, which were indeed present, but not necessarily acting as exciting causes. In support of this assertion, it is sufficient to recall the fact that many healthy persons are infested with tape-worms and present no symptoms; and also, that many persons suffering from various diseases have tape-worms, and these more than other persons are apt to expel them, and thus mislead.

It may, nevertheless, be readily granted that those who have a delicate or irritable mucous lining to their intestines, or who are of a nervous temperament, and abnormally liable to reflex excitement, do suffer some, perhaps many, of the symptoms here recounted, and that in stronger persons the same may happen if the worms are very numerous. But it is worth remembering, that paroxysmal maladies, such as convulsions, mania, &c., are peculiarly liable to give rise to errors in reasoning as to their causes, so that very rarely could it be affirmed that they were caused by a tape-worm when their cessation coincided in time with its expulsion.

In some cases, proportionally few in number, when abscesses have formed in connexion with an obstruction of the intestine, a tape-worm has escaped from the opening, and may have been partly, or perhaps solely, the cause of such obstruction and abscess.

There is another fortunately rare, but grave consequence of the presence of a tape-worm; it may give rise to the development of the

Cysticercus cellulosæ in the tissues or organs of its bearer, and thus even destroy life. This may conceivably take place when, as a consequence of violent vomiting, some of the ripe joints are carried up into the stomach, where the digestive fluids might set the embryo free; or in the case of children or dirty people, by conveying the escaped segments or free ova, upon the hands or with the food into the mouth, and thence into the stomach.

Diagnosis.—When a patient presents such a conjunction of symptoms as, in the absence of other indications, excites a suspicion of tape-worm, its presence can only be ascertained by an inspection of the stools. The ripe segments (Fig. 5) or the ova, (Fig. 8 *b*) will with a little care almost certainly be found in the fæces, and from them the species may be determined with sufficient exactitude for the requirements of the physician.

Etiology.—The exciting cause of the disease is manifestly the worm, a foreign irritating body in the intestine. The favouring conditions are the adult age, possibly the female sex, certainly some occupations, such as those of the cook or the butcher, the habit of eating raw or underdone pork, ham, sausage, &c., and a residence in Europe, India, Algeria, North America, and probably wherever the pig is domesticated.

Pathology.—Leuckart has shown, by observations on the dog, that local congestions of the mucous membrane, separation of the epithelium, and even minute superficial sores, may result directly from the activity of a tape-worm. If it be admitted that *T. solium* may cause similar local changes in man, there is no difficulty in connecting the deranged functions of the alimentary canal with the worm as their cause, if we grant either an exceptional delicacy of the bearer, or an unusual number of worms. The remote functional disorders present no more difficulty, if pre-existing abnormal proclivity to reflex movements be granted.

Treatment.—The indications for treatment follow in the clearest manner from the foregoing. The worm as exciting cause must be got rid of, and the effects then commonly subside; but should they persist for a time, they can be successfully met by suitable diet and the treatment for irritation of the intestines.

An immense number of substances have, at various times, enjoyed a reputation for the possession of anthelmintic powers, too often without any accurate distinction of the kind of worm, so that with the rise of a more accurate diagnosis, as well as, perhaps, of a more critical spirit in modern times, the number of accepted remedies for tape-worm has rather diminished, and a general demand has arisen for a re-examination of the claims of most of the reputed agents.

THE MALE SHIELD-FERN (*Aspidium filix mas*) is perhaps the oldest and most widely known vermifuge, and of late has grown into much favour, especially in this country.

The dose is from 60 to 100 grs. of the powder of the dried rhizome, or from ʒj. to ʒij. of the liquid extract, given upon an empty stomach,

nsiders the old and dry bark almost inert. He prepares the
by spare diet and aperient medicines, and then gives a pint
ecoction much like that of the British Pharmacopœia (equal
. of bark) in three doses, at short intervals, early in the morning.
nmeister uses a still stronger decoction, and gives a quantity
o 4 oz. of the pomegranate bark, with 20 grains of the ethereal
of male fern added. The German authorities generally employ
ul, not to say violent measures for the expulsion of tape-worm,
w far this may be due to the greater resistance which some
present, is unfortunately not yet certain.

sso—the flowers and tops of *Brayera anthelmintica.*—In doses
½ oz. or more it is a quick and good anthelmintic, much used
ssinia for the species of tape-worm there prevalent. It is not
used in Europe, perhaps on account of its cost, of the difficulty
aining it, and of the inconvenient form in which it is usually
istered.

nala, from the fruit of the *Rotleria tinctoria, oil of turpentine,*
number of other agents have been recommended, but it is not
ble to notice them here. Some rare instances occur in practice,
ich treatment by any or all of the above-mentioned drugs fails
el the worm so as to prevent its recurrence, which takes place
ly whenever the head and neck remain attached. Some cases
are recorded in which even the expulsion of the greater part of
nd is not effected; and this not only when moderate doses have
sed, but even after elaborate preparation, vigorous treatment, and
bsequent purgation such as Wawruch and other German autho-
ave advised. No very satisfactory explanation can be offered
s singular power of resistance occasionally met with; but in
ce of the admitted failure of violent irritating remedies, it would
rudent in such cases to continue moderate doses of male fern or
ranate for longer periods of time, in conjunction with rigid
lactic rules, to prevent the possibility of reinfection,

vention.—Each person can secure himself against *Tænia solium*
ting only such pork, ham, sausages, &c. as are well cooked:

TÆNIA MEDIO-CANELLATA (Kückenmeister).

Description.—This worm was formerly held to be an unarmed variety of *T. solium,* but Kückenmeister and Leuckart have recently established its specific distinctness both by observation and experiment. It has a general resemblance to, but is larger and firmer in texture than, *T. solium;* not only does the whole band (Strobila, Fig. 9) commonly attain a greater length, but the segments are more numerous, and larger in all their dimensions. The unripe ones are broader than long, the ripe ones longer than broad. The contained uterus (Fig. 10) is more
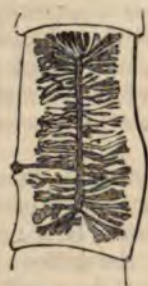
FIG. 10.—Ripe joint of *T. medio-canellata.* (Leuckart.)

FIG. 11.—Head of *T. medio-canellata,* magnified. (Davaine.)

FIG. 9.—*Tænia medio canellata,* natural size. (Davaine.)

FIG. 12.—Ovum of *T. medio-canellata.* (Davaine.)

FIG. 13.—*Cysticercus T. medio-canellatæ,* natural size and position. (Leuckart.)

finely divided than in *T. solium,* having from 20 to 35 branches on each side. The common sexual aperture is placed alternately on either border, nearer to the posterior margin than in *T. solium.* The head is large (Fig. 11), measuring about $\frac{1}{12}$ in. (Davaine); has neither rostellum nor coronet of hooks; is furnished with four very powerful and prominent suckers; and, according to Leuckart, a fifth smaller one in the usual position of the rostellum (Fig. 15). Kückenmeister also figures a central canal connected with the water-vascular system.

The eggs (Fig. 12) resemble those of *T. solium* except that they are
ore oval in outline : they measure about $\frac{1}{700}$ in. by $\frac{1}{850}$ in.

The larval form, or *Cysticercus tæniæ medio-canellatæ*
(Figs. 13, 14, and 15), infests the flesh and organs of the
ox, a fact which at once points out the chief difference
between its life history and that of *T. solium*.   *T. medio-
canellata* abounds in Abyssinia and South Africa, and
is also common in Europe : it was, until the recent re-
searches of Dr. Cobbold, thought to be more common in
continental states than in this country ; but it is now
known to occur almost, if not quite, as frequently amongst
us as *T. solium* does.

Fig. 14.—*Cysticercus T. medio-canellata.* Head inverted. Magnified. (Leuckart.)

Notwithstanding its being unarmed, the great strength of its suckers
able the head to hold on with even greater tenacity than the *T.
lium*, so that it is more difficult to
pel, and it is believed to excite
ore marked symptoms ; but as the
rval form does not, so far as is at
esent known, infest man, it is less
ngerous to life.

The terminal joints separate spon-
aneously from the parent chain, and
ften creep out of the anus irre-
pective of the passage of fæces ;
s a rule having first permitted at
ast a portion of their contained
va to escape by rupture into the
testine.

So far as is at present known, its
eatment is the same as that for
T. solium, and its prevention consists
the avoidance of raw or underdone
eef.

The three following *Tæniæ* are
laced by Leuckart in a separate
roup, of which the larvæ are dis-
nguished by having comparatively
nall caudal vesicles, and are met

Fig. 15.—Head of *Cysticercus T. medio-canel-
latæ*, more highly magnified, showing central
sucker. (Leuckart.)

ith only in cold-blooded, generally invertebrate animals.   Those
curring in man are minute, and have been so rarely met with, at
ast in Europe, as to be of comparatively little clinical importance.

TÆNIA NANA (Von Siebold)

scarcely an inch long, and about $\frac{1}{70}$ in. wide at its broadest part.
ead globular, with an oval rostellum bearing a single row of 22
24 very minute hooks, and four rounded suckers.   Eggs globular,

$\frac{1}{640}$ in. Found by Bilharz in great numbers in the duodenum of natives of Egypt. Its migrations and metamorphoses are unknown.

TÆNIA FLAVO-PUNCTATA (Weinland).

The adult attains a foot in length. The joints of the anterior half of the chain are marked by a distinct yellow spot, the receptaculum semini, which is absent in the following segments. Head unknown. The egg measures $\frac{1}{425}$ in. Met with but once, in a healthy infant in North America. Life history unknown.

TÆNIA ELLIPTICA (Batsch).

The adult worm attains a length of 6 in. to 8 in., head very minute, measuring $\frac{1}{80}$ in., rostellum cylindrical, furnished with three or four rows of hooklets. Terminal segments three or four times as long as broad. Sexual apertures double, one on each margin of segment. Eggs measure $\frac{1}{500}$ in. It infests normally the intestine of the cat, and only very exceptionally has been found in man. Its life history is unknown.

Here I venture to add an abstract of a case (*Med. Times and Gazette*, p. 598, 1856) which suggests the possible addition of still another species of *Tænia* to the above list. A girl aged nine years, suffering from disordered digestion and impaired nutrition, passed with the fæces for more than fifteen months consecutively numerous oval ova

(Fig. 16), measuring about $\frac{1}{650}$ in. by $\frac{1}{500}$ in. and containing a globular embryo, furnished with three pairs of hooklets. These eggs differed so much from those of any other tape-worm then known to me, that I referred them to an undescribed species of *Tænia;* but whether this may ultimately prove to be correct or not, the view receives some support from the fact, that although the girl during the whole of that time was under observation

Fig. 16. — Ovum of *Tænia* of uncertain species.

as a hospital patient, was treated vigorously and repeatedly with male fern, kousso, pomegranate bark, turpentine, and various cathartics, and the stools carefully watched, yet no tape-worm joints were ever found, although the ova continued to be expelled in undiminished numbers. It is very difficult to suppose that the child harboured a *T. medio-canellata* which, although sexually mature, passed no joints, yet this is the only *Tænia* known to me of which the ova have even a passing resemblance to those found in this case. It seems more probable that the tape-worm was one which normally expels its ova without casting off joints of such dimensions, or in such a condition, as to be recognisable in the stools on a careful search.

In this case the functional disorder subsided shortly after treatment began, but as the ova continued to escape, it could not have been

ed by the parasite or parasites. Ultimately the patient ceased to
nd, but to the last her fæces contained the same ova.

he two remaining tape-worms of man belong to the family
riocephalidæ, of which the adult forms infest chiefly cold-blooded
ebrate animals.

BOTHRIOCEPHALUS LATUS (Bremser).

escription.—This is the largest tape-worm known to inhabit man ;
ommonly reaches a length of 17 to 26 feet, and sometimes 60 feet

FIG. 17. — *Bothriocephalus latus,* natural size. (Davaine.)

FIG. 18.—Head of *Bothriocephalus latus,* magnified. (Davaine.)

FIG. 19.—Ova of *Bothriocephalus latus :* a with contained yolk; b empty shell. (Leuckart.)

more (Fig. 17). The head (Fig. 18) is unarmed, oblong, or club-
ped, it measures $\frac{1}{10}$ in. in length by $\frac{1}{15}$ in. in breadth, has a
ply-grooved longitudinal sucker on each side, and passes gradually
a short thread-like neck. The joints are broader than long, the
est being $\frac{1}{4}$ in. in length, by $\frac{1}{2}$ in. or even more, in breadth ; towards
posterior end of the chain they increase in length and diminish in

breadth, assuming thus a more square form; they are thicker in the middle than at their margins, from the presence there of the sexual organs, which form a rosette-shaped patch in the centre of which the sexual apertures are placed. The eggs (Fig. 19) are oval, $\frac{1}{310}$ in. by $\frac{1}{570}$ in.; have a firm, brownish, structureless shell, with an operculum at one end. While yet within the uterus they present no trace of embryo in their interior.

Life History.—The ova escape by rupture of the ripe joints, and probably in part also through the oviduct, into the intestine before the joints separate; these are expelled with the stools at rather long intervals; not singly, as is often the case with *T. solium* and *T. medio-canellata*, but in short chains of 2 to 4 ft. in length. The ovum after a prolonged sojourn in water develops a ciliated embryo, which escapes through the aperture in the shell by forcing open the lid, and is furnished with three pairs of hooklets. On analogical grounds it is very probable that it then enters into the body of some aquatic animal, possibly a fish, but probably a mollusc, and then assumes the larval form, which is at present unknown. The intermediate bearer is probably eaten by man, and the larva assumes the adult form in his intestine. *B. latus* usually occurs several together; it has a somewhat limited geographical distribution, not having been found beyond the limits of Europe; in some countries of which only is it indigenous. It is common in the western cantons of Switzerland, North-western Russia, Sweden, Poland, Holland, Belgium, and Eastern Prussia; it is less often met with in other parts of Germany, and has occasionally been imported into Britain. Low-lying damp regions near the borders of seas and lakes are those in which it is most often abundant. It is found in persons of all ages and sexes, and in those countries where it is most frequent even children at the breast are not free from it.

The *Symptoms* do not differ from those caused by the species of *Tænie*. Its presence may be detected by an examination of the stools. It may be expelled by the same drugs as are employed in the treatment of other tape-worms, and it is said to be less difficult to dislodge, perhaps on account of the feeble development of its suckers. No precise knowledge has yet been attained of the measures to be taken to avoid it, but the general rule of carefully cooking all foods and of drinking only pure water would be likely to succeed even in those countries where it most abounds.

BOTHRIOCEPHALUS CORDATUS (Leuckart).

A recently discovered, much smaller worm, found only in North Greenland in men and dogs. It is known by its caudate head and the absence of a neck.

Order *NEMATODA.*

ted, slender, often thread-like worms, not distinctly jointed,
ed with appendages ; with a separated alimentary canal, a
mouth, an anus (*Gordius* excepted) near
l extremity, opening on the ventral aspect.
gument is marked by two lateral longitu-
ds, and often by a dorsal and a ventral
he former are embedded the nerves with
glia, and the excretory tubes, which open
rface about the level of the pharynx.   The
perture is placed near the central region
dy, that of the male near the anus, and
l with it : it is furnished with retractile
usually two or more.   The male is smaller
female.   The development is direct and
amorphosis inconspicuous ; so that the
as the general aspect of a nematode worm.
r is rich in species, and furnishes as many
as all the other Helminthoids put together.
st invertebrata as well as vertebrata, and
s escape their invasion,

IS LUMBRICOIDES (Linnæus).   Common
round-worm.

tion.—A large nematode worm, during
reddish or brownish tinge, and of a firm,
xture (Fig. 20).   The female reaches 15 in.
by ½ in. to ¼ in. in breadth ; and the male
¼ in. (Leuckart).[1]   The cylindrical body,
by a cuticular layer and marked by fine
e rugæ, tapers towards both ends, but more
owards the head ; in which is placed the
mouth, surrounded by three nearly equal
t muscular and tactile lips (Fig. 21), each
high as broad, and marked off at its base
inct groove.   The inner surface of each
set with about two hundred very minute
pic teeth,   The triangular mouth conducts
scular œsophagus, and this to a simple,
traight intestine, without distinction of
The lateral longitudinal bands, much
inct than the median, divide the muscular
nearly equal areas, and give attachment
fibres, as well as support the nerves and
tubes.   The caudal extremity, short and

e measurements exceed those given by Davaine.

O

FIG. 20.—Adult female, *Ascaris lumbricoides*, natural size.   (Leuckart.)

conical, terminates in a point, and in the male curves strongly towards the ventral aspect, on which is seen the cloacal aperture with two often projecting spiculæ (Fig. 22). These are connected with a short, ejaculatory duct, which is continuous with a seminal vesicle, and a single long, tortuous, tubular testis; the whole male generative organ forming a tube eight times the length of the animal. The vulva in adult females opens about the junction of the anterior and middle third of the body, it conducts to a short vagina, this to a uterus, which soon divides into two long horns, directed backwards; each of these leads to a short oviduct, which serves also as a receptaculum seminis, and thence to a very long, tortuous, tapering ovary. The female generative tubes are eleven times the length of the adult animal.

Fig. 21.—Head of *Ascaris lumbricoides*, magnified. (Davaine.)

The ova are oval in form, and have a thick, firm, elastic, brownish shell, the surface of which is generally nodulated. No commencement of development is seen in their interior when deposited. They measure $\frac{1}{840}$ in. by $\frac{1}{410}$ in. (Fig. 23, *a* and *b*).

Life History.—So fertile is the round-worm, that, at a moderate calculation, its yearly production of ova may be taken at 60,000,000, so that over 160,000 are daily discharged into the intestine of its bearer by one adult female worm. As, however, several are often present together, it is easy to understand that the stools of an infested person are so thickly strewn with the eggs as to make their discovery by the microscope an easy matter.

Although the migrations of the embryo of *Ascaris lumbricoides*, and the true history of its development, are not yet ascertained with sufficient ex-

Fig. 22.—Caudal extremity of male *A. lumbricoides*, magnified. (Leuckart.)

actitude, the labours of Schubert, Verloren, Davaine, Leuckart, and others, permit the following history to be given as an approximately correct statement of the facts. The ova deposited with the fæces very slowly develop an embryo in damp earth or water. The process may be complete in a month if artificial warmth be applied, but in nature it usually requires from five to eight months, and it may be delayed for a year or two by cold or dryness. Neither frost nor complete desiccation, however, kills the embryo, and the contained ova of dried females develop under suitable conditions. The ova do not normally hatch in a free state; Davaine has preserved them in water for five years

Fig. 23.—Ova of *A. lumbricoides*, from the stools: *a* recently deposited; *b* longer delayed in the stools. Shell tuberculated.

without any visible change in the embryo, or spontaneous escape from the shell. In this stage the embryo has the general aspect of a nematode worm, with an alimentary canal, a commencing generative system,

and a terminal boring, embryonic tooth. The next stage of their development is not known. Davaine maintains that the ova with their contained embryos are swallowed with impure water, and develop directly into the adult form if received into the intestine of a suitable bearer. But direct experiments do not support this view; dogs, rabbits, oxen, pigs, and men have been fed with large numbers of the ova of *A. lumbricoides* containing living embryos without any infection resulting. Similar experiments conducted upon horses, dogs, and cats with the ova of their peculiar round-worms have had similar negative results, and it seems indeed almost certain that infection does not take place by a direct transference of the embryo-holding ova into the alimentary canal of the definitive bearer. It may be said also with some confidence that the embryos do not escape from the ova to enjoy a free existence for a time. On analogical and other grounds it is a far more probable view that the ovum is taken up in some way by an invertebrate intermediate bearer, perhaps a worm, or the larva of an insect, and in it the embryo passes through a necessary portion of its metamorphosis, and then enters the stomach of its future host in some passive mode with food or drink.

Ascaris lumbricoides infests also the pig [1] and the ox: it is found in man all over the known world, but more abundantly in some countries than in others. In the Southern States of North America, especially among the negroes, it attacks almost every one, young and old. In the West India Islands, Brazil, Finland, Greenland, in parts of Holland, Germany, and France, it is also very frequently met with. The rural population suffer more than the dwellers in towns, and the inhabitants of low and damp localities more than those who enjoy higher and drier abodes. The poor, the young—excluding infants at the breast—the insane, and the dirty, are peculiarly liable to be infested. In certain regions it has occasionally prevailed so much for a time as to produce a kind of endemic malady.

The round-worm normally inhabits the small intestine, and there is some ground for the opinion that, unless a reinfection occurs, it escapes after some months. There can, however, be no doubt that it spontaneously wanders towards the external apertures under certain conditions which are not well known, sometimes passing through the anus, the mouth, the nose, often with severe purging, vomiting, or sneezing. After death, also, this migration is not uncommon, and is probably induced by a deficiency of food, or the presence of some conditions unsuitable for the welfare of the worm; but whatever induces it, it results in placing the worm occasionally in remote and singular localities, both during the lifetime and after the death of the sufferer. It creeps sometimes into the gall duct, gall bladder, or hepatic duct, more rarely into the pancreatic duct, and may give rise here to serious structural changes: it passes sometimes through an ulcer or other abnormal opening in the intestinal wall, and then is

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[1] Leuckart considers this species identical with *A. Suilla.*

found after death in the peritoneal cavity, accompanied or not with the signs of peritonitis, according as it may have migrated during life or after death ; it escapes sometimes with other intestinal contents from abscesses or fistulæ in the abdominal walls, and appears, indeed, in some such instances to have caused the local disease. It has so marked a tendency to creep into small apertures, that several instances are recorded of its becoming fixed in the eyes of buttons and other similar small rings which had been swallowed by the patient, and this habit has even suggested the swallowing of such rings to act as worm traps. This migratory instinct has occasionally led the round-worm along fistulous channels to still more remote cavities or organs ; for example, to the pleural sac, the spleen, the kidney, the bladder, the muscles of the loin or neck, the spinal cord, the lung, the glottis, the trachea, and the Eustachian tube.

In the more favoured countries, usually from one to five worms are met with together, but often many more are present ; cases are recorded in which various numbers, from 200 to 2,500, have been expelled from one person within a few months, and 1,000 were found present together in the intestine of an idiot by Cruveilhier.

Symptoms.—The round-worm is one of the most frequently met with, and is clinically more important than any other human intestinal worm. When it is present in moderate numbers, and occupies its normal position in the small intestines in a person otherwise healthy, there are often no discoverable disorders of structure or function. When present in greater numbers, or infesting a delicate person, it is accompanied by the symptoms of irritation of the lining membrane of the alimentary canal, and by consequent impaired nutrition and reflex phenomena. Thus it may be attended with pain in the abdomen, especially in the umbilical region, nausea, impaired or variable appetite, mucous stools, and tumid abdomen. Sometimes, also, pallor of the surface, dilated pupils, swollen eyelids, squinting, irritation of the nostrils, grinding of the teeth during sleep, &c. : indeed, all the allied symptoms which have been attributed to tape-worm. But these are by no means constant effects of the presence of round-worms in the intestine, nor are they peculiar to their irritation. They may be absent when worms are present in considerable numbers ; and may be present when no worms infest the patient ; or present with the worms, but not caused by them. They have, therefore, little or no diagnostic value. Sometimes, however, especially when the intestine contained these worms in very large numbers, they have caused grave local irritation as well as constitutional disturbance, and then post-mortem examination has shown evidences of local superficial congestions and inflammation so closely related to them in extent or position, as to leave no doubt of their causal relation. Thus cases are recorded where numerous round-worms, cohering to each other, gave rise to fatal obstructions and inflammation of the intestines, and others in which they have excited serious and even fatal convulsions in susceptible persons. Although in these latter cases the reflex symptoms are probably in no

essential point different from those caused by other irritations, it is important to trace them to the worms, if it can be done, because of the comparative facility with which the exciting cause can be removed. In the rarer cases in which the round-worm wanders during life into distant cavities, organs, or passages, the disorders they induce vary with the parts visited, and may be of great severity, or even terminate fatally.

Diagnosis.—When, for any reason, a patient is suspected to harbour round-worms, it has been a not unfrequent practice to employ the usual treatment for their expulsion—often a rather vigorous one—as a means of diagnosis; and should no worms be passed, it has been assumed that none were present: thus submitting the patient to treatment before the need for it is made out, and assuming, somewhat hastily, that the recognised treatment may be relied upon.

An easy and satisfactory method of diagnosis consists in the microscopic examination of the stools, in which, if the suspected person harbours a mature female, the ova[1] are readily seen. I published a case in the *Medical Times and Gazette* for June 14th, 1856, which so well illustrates the value of this method for diagnosis, and its bearing on treatment, that I venture to give here the following summary of it:—

A girl, aged twelve years, had passed two round-worms before she came under observation, and had complained for six weeks of abdominal pains and disordered digestion. For convenience of observation she was admitted into hospital Feb. 14th, 1855; her stools then contained ova of *Ascaris lumbricoides* (Fig. 23). After nine days, during which she was treated by a mixture of bicarbonate of soda and infusion of quassia, with rest and good diet, she declared herself well, but had passed no worms. For ten days more she was treated by oil of male fern and castor oil, followed by scammony, without effect. For a further period of ten days she took infusions of quassia and senna, also without result. For five weeks more she was given turpentine and castor oil, or turpentine alone, at weekly intervals; and about the third or fourth day after each dose, except the last, she passed one or two worms, generally, but not always, motionless. The ova were still abundant in the stools, but the treatment failing to expel any more worms, she was given *Dolichos pruriens* for four days, until it caused nausea, when it was omitted: but for twelve days more she expelled occasionally one or two worms with the stools. The *Dolichos pruriens* was then repeated for eight days, and again omitted; after which she passed, in the following fortnight, three more worms. The ova were then found to be absent from the stools, and she was discharged. While under treatment she passed, in all, seventeen round-worms; but during the last three months and a half she was in perfect

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[1] It is curious to note that these ova have been described as cholera corpuscles (*Lancet*, 1849, p. 532); and more recently as "choleraphyton," in the *Deutsche Klinik*, 1867.

health, and would have been discharged but for the observation of the ova in the fæces.

Davaine drew attention to the value of this method of diagnosis in 1857 (*Comptes Rendus Soc. Biologie,* 2ᵉ Série, t. iv. p. 188); and Leuckart says (*Die menschlichen Parisiten,* &c., B. ii. p. 251, 1867), " In the microscopic examination of the fæces we possess a means to determine the presence of the round-worm, which is as easy as it is sure; if it were more generally practised, many errors of diagnosis, and many useless, if not injurious treatments, would be avoided."

The *Etiology* and *Pathology* of the disorders induced by round-worm have appeared on the surface during the previous observations.

Treatment.—The indications are to relieve the irritation of the alimentary canal, to improve the general nutrition where that has suffered, but above all things to expel the worms. Many of the substances which have obtained a reputation as anthelmintics have been much used for round-worm, but we have as yet no sufficiently exact knowledge of their action upon the different species of intestinal worms to enable us to estimate their true clinical value in the treatment of *Ascaris lumbricoides.* There exists, however, a very general concurrence of opinion, which I believe to be well founded, in favour of the use of santonica or worm-seed, the unexpanded flower-head of an undetermined species of *Artemisia,* as well as of its active principle, santonin. The dose of the worm-seed is from 60 to 120 grains, but it is not much used on account of its inconvenient form; that of santonin, which is more used, is from one to three grains twice daily to a child, and from three to six grains for an adult. After a short course of this medicine, an aperient may be given with advantage. It is apt to produce a singular although but temporary perversion of vision if given in too large doses, or for too long a time, objects seeming to be yellow, blue, or green. The urine also may be tinged red after its use. Violent cathartics do not deserve much confidence, nor are the drugs employed for tape-worm (except, perhaps, turpentine) to be trusted to. *Dolichos pruriens* would seem to be worthy of further trial in some cases where santonin is not available, but of the numerous other substances which have been at times recommended for the treatment of *A. lumbricoides,* it is unnecessary to say more here.

The *Prevention* of *Ascaris lumbricoides* cannot be so confidently treated of as was that of *T. solium,* because we are not certain how it enters our bodies; but whether we hold with Leuckart that an intermediate bearer is essential, or with Davaine that it is not, and that we drink the ova in impure water, in all probability the careful cooking of all our foods and drinks would prove a good protection even in those countries and districts in which this pest most abounds. It is not, however, probable that well-filtered water could convey the infection.

ASCARIS MYSTAX (Zeder)

the common round-worm of the cat, and is identical with *Ascaris*
rginata of the dog (Schneider).

Description.—It is smaller and more slender than *A. lumbricoides*,
two small lateral, cuticular, wing-like appendages near the head.
: vulva in the adult female occurs about one-fourth of the whole
gth from the head. In man it has only been found parasitic in
ee trustworthy instances, which are recorded by Bellingham,
bold, and Leuckart.

OXYURIS VERMICULARIS (Bremser), (Common seat-worm).

Description.—A small whitish fusiform worm, the female attaining
n. in length by $\frac{1}{16}$ in. in thickness, and the male about $\frac{1}{6}$ in. in length
$\frac{1}{100}$ in. in thickness (Fig. 24).
: head (Fig. 24 *b, d*) is furnished
h three inconspicuous lips around
rminal mouth, and an elongated
icular expansion of the cuticular
:r on its dorsal and ventral as-
:ts. The œsophagus is continuous,
h a muscular stomach containing
:e teeth, and then follows a simple
:stine. The surface is marked by
transverse rugæ, and the lateral
gitudinal bands form a slight an-
ar projection. The female has a
g. awl-shaped, caudal extremity
g. 24 *c*); the vulva is situated
at the junction of the anterior
middle thirds of the body, and
ducts to a vagina, a bifid uterus,
this to two tubular ovaries. The
e has a blunted tail end furnished
h six pairs of papillæ, and a

FIG. 24.—*Oxyuris Vermicularis. a* Natural
si:e *b* Head, magnified. *c* Tail, magnified
d Head, more magnified. (Davaine.)

gle spiculum communicating with the anal aperture. The eggs
g. 25) are oval but flattened on one surface, measure $\frac{1}{1100}$ in. by $\frac{1}{400}$ in.,
tain at the time of deposition a developing embryo, and have a
shell consisting of three layers, one of which is absent at one
e, so as to facilitate the escape of the embryo. A moderate estimate
ws 10,000 to 12,000 ripe ova for the uterus of a single female.

Life History.—The seat-worm, like the round-worm, is found all
r the world, and is perhaps even more frequently met with. It is
to abound particularly in Egypt and in Greenland. It normally
bits the colon of man only, especially the neighbourhood of the
um, and is commonly found in large numbers, the males fewer
the females, and it often migrates spontaneously through the

anus.   The ova are discharged into the intestine of the infested person, and there undergo a further development, so that at the period of their escape with the stools they usually contain a distinctly formed embryo.   The frequent spontaneous migrations of the ripe female also often lead to the deposition of the ova upon the skin and hair in the neighbourhood of the anus.

The ova deposited with the stools rather rapidly develop under favourable conditions, especially moisture and the warmth of the sun; they are not killed by extreme cold or by desiccation, but a few days' delay in water kills them outright, and under ordinary circumstances they die in a few weeks unless their progress has been arrested by cold or dryness.   It does not seem that they hatch in the free state.

Fig. 25.—Ovum of *Oxyuris vermicularis,* from the faeces. (Leuckart.)

Kückenmeister and Vix conceive that all the transformations from the embryo to the adult form take place within the intestine of the infested person without any necessary migration, and at first sight this view seems to receive support from the fact that large numbers of seat-worms are commonly found together, and that various grades of development are there met with.   This view, however, is out of accord with the general law of development in parasitic animals, and does not suffice to explain the known facts. Leuckart insists that the emigration of the embryo is a necessary condition of its future development, and has indeed almost proved the correctness of this view by observation and experiment, as well as by powerful arguments.   His view is, that the ova deposited with the faeces are abundantly and widely scattered in the dry state by winds and other agencies, and then are taken into our stomachs upon uncooked fruits and vegetables and in various other conceivable modes; there, exposed to the digestive fluids, the embryos escape, are carried down into the colon, and attain the adult form probably in about two weeks. A sort of self-infection frequently may take place also; in persons already infested, it is easy to see how the ova upon the skin and hairs near the anus may be conveyed to the mouth by the fingers, after scratching to allay the violent irritation which these small pests produce; and in other modes the eggs may find their way into the stomach from the soiled bed-clothes or personal linen.   These views explain some long-known facts which are not otherwise so easily understood; for instance, the great length of time during which some persons suffer from seat-worms, and the liability to relapses notwithstanding repeated treatment: the frequency with which these worms are found inhabiting many members of one family or household, the greater liability of children, of dirty or insane people, and of persons who often eat uncooked fruit and vegetables, as well as the immunity of infants at the breast.

Symptoms.—When only a few seat-worms are present, they give rise to no inconvenience, and are usually only accidentally discovered in the stools.   When they are more numerous, or the patient is more sensitive, they cause an itching or tickling in the anus and its neigh-

bourhood, which is sometimes intolerable to the sufferer, especially at a certain hour in the evening. In females it is peculiarly distressing, from the habit which the worm has of wandering into the vagina; but in both sexes inordinate sexual excitement sometimes is produced. Although there is sometimes evidence of local irritation in the shape of excess of mucus in the fæces and punctiform redness around the anus, the cases of severe convulsion and other nervous disorders which have been referred to the action of seat-worms must be received with much caution.

Diagnosis.—Inspection of the stools will discover the worms; and a microscopic examination will show the ova.

Treatment.—Probably any infected person who adopted the requisite precautions against reinfection from himself or others would get well in a few weeks without treatment by drugs,[1] but this period would be shortened by the use of aperients and occasionally injections into the rectum of cold water, turpentine and castor-oil with gruel, and of preparations of wormwood, quassia, assafœtida, santonin, &c. Frequent external applications of mercurial or other ointments and lotions likely to kill the embryos might be employed also.

Prevention.—From the foregoing history, it may be learned that a sufferer from seat-worms should avoid touching the neighbourhood of the anus, should be scrupulously clean[2] in person and in clothing; that persons not yet infested should avoid close personal contact, especially in bed, with those who harbour the worms, and should also adopt the caution of eating only well-cooked food.

Family STRONGYLIDES.

DOCHMIUS DUODENALIS (Leuckart).

This minute but dangerous parasite was discovered by Dubini in 1838, in Northern Italy; its zoological position is scarcely yet settled, but its close affinity to the genus *Dochmius* of Dujardin has been shown by Molin and Leuckart.

Description.—It is a small somewhat cylindrical worm: the females measure $\frac{7}{10}$ in. and the males $\frac{4}{10}$ in. in length (Fig. 26.) The terminal mouth is surrounded by a dilated capsule directed obliquely backwards, and furnished with four large teeth on its longer or ventral border, and with four smaller ones on the opposite or dorsal margin (Fig. 27). The bursa of the male is complex, the spicula two in number. The vulva of the female is placed a little behind the centre. The eggs are oval, measure $\frac{1}{110}$ in. by $\frac{1}{1080}$ in., and when deposited contain a yelk in process of cleavage.

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[1] This appears to be a daring statement in the face of past experience, but its probability is measured by the evidence for the life history here given.

[2] The common Hindoo custom of washing after every act of defæcation is worthy of more frequent imitation in this country.

We know as yet but a part of its life history by direct observation, and infer the remainder from that of the better known and very closely allied *D. trigonocephalus* of the dog. The egg, after escaping with the stools, under favourable conditions hatches in a few days, and the embryo enjoys a free existence for a time in mud and muddy water. It is taken into our stomachs by drinking impure water without the intervention of any intermediate bearer, and there it grows and develops to some extent before it passes on into the duodenum or jejunum, where the adult form is assumed. It then attaches itself by its powerfully armed mouth to the villi of the mucous membrane, and sucks the blood of its host. Sometimes, under conditions not yet explained, it becomes encysted between the mucous and muscular coats of the gut. It occurs in warm countries only, has been found in Italy (Dubini), Brazil (Wucherer), and in Egypt (Pruner, Bilharz, Griesinger), where it is a very frequent and dangerous pest, infesting about one-fourth of the entire population. It is present in large numbers together, often by hundreds, sometimes by thousands, and then may cause frequent and dangerous hæmorrhages into the bowels,

Fig. 26.—Male and female *Dochmius duodenalis*, magnified. (Leuckart.)

followed by an anæmic condition, which is often fatal, and to which the name of Egyptian chlorosis had been given before Griesinger pointed out its true nature.

Doubtless its ova might be found in the stools of infested persons, but of the treatment which should follow a diagnosis so established little can be said, except that Griesinger recommends turpentine, and that santonin and such other substances as are believed to expel nematode worms should be tried. Care should also be taken to consume only pure water or drinks which have been boiled, so as to avoid re-infection, and the patient might then be fairly expected to outlive the worm.

Fig. 27.—Head of *Dochmius duodenalis*, magnified, showing the armature of the mouth capsule. (Leuckart.)

Although to the practitioner in Britain this parasite is not of practical import, it seems so probable that it may be found in India or some of the tropical British colonies, that I have ventured to include it here.

Family TRICHOTRACHELIDES.

Trichocephalus dispar (Rudolphi).

Description.—The female measures about $1\frac{9}{10}$ in. the male about $1\frac{6}{8}$ in. in length. The anterior three-fifths of the body are threadlike, measuring $\frac{1}{100}$ in. only in thickness, and bear a simple terminal mouth without papillæ. The posterior two-fifths, about $\frac{1}{12}$ in. in thickness, contain the generative organs and the intestinal canal; in the male it is spirally coiled, in the female slightly curved (Fig. 28). The caudal extremity is rounded off in the male, and bears a single blunt spiculum in a tubular protrusile sheath which is furnished with teeth. The vulva in the female opens about the level of the stomach into a vagina, the walls of which are furnished with teeth, and often prolapse. The large uterus contains thousands of eggs, which are elliptical in form, and have a nipple-shaped projection at each end They measure $\frac{1}{140}$ in. by $\frac{1}{1120}$ in. (Fig. 29), and have a firm brownish yellow shell, wanting at each pole, so as to leave an aperture which is closed by a firm transparent nipple-shaped plug. As found in the stools the yelk shows no trace of commencing development.
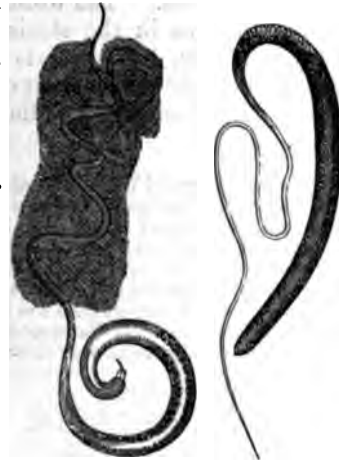
FIG. 28.—Male and female *Trichocephalus dispar*, magnified. (Leuckart.)

Life History. — The *Trichocephalus crenatus* of the pig, and also that found in some monkeys, is probably the same as our *T. dispar.* It is met with in most, if not in all, European countries; in Syria, Egypt, and North America; it abounds in Italy, and in some Eastern lands; but is said to be comparatively rare in Copenhagen and in London (Cobbold). It does not generally occur in large numbers together, although sometimes hundreds have been found. The head of the colon is its chosen residence, but occasionally it is met with in the intestines near. During the life of its host, it attaches itself by thrusting its long whip-like neck into the mucous membrane. The ova deposited with the stools, like those of *Ascaris lumbricoides*, very slowly develop normally in damp earth or water, so that in warm weather and under favourable conditions the embryo is formed in about four or five months; but in cold weather or exposed to temporary drought it
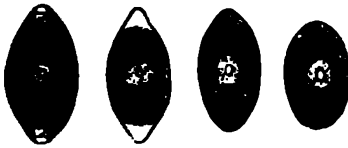
FIG. 29.—Ovum of *T. dispar.*

requires a year and a half or more. In this state the embryo
remains, and neither develops further nor leaves the shell to become
free. (Davaine has preserved them alive in this stage for four years.)
From Leuckart's experiments upon the *Trichocephali* of sheep and
pigs, it is highly probable that no intermediate bearer intervenes,
but that we swallow the ova with their contained embryos in some
accidental manner, as dust upon uncooked fruit, vegetables, &c. &c.
and that the embryos escape into our stomachs after partial digestion
of the shells, develop somewhat, and then travel onwards to the
colon, where they become sexually mature in four or five weeks.

No symptoms are known to be caused by *T. dispar*, although some
writers have attributed severe reflex disorders to them when present
in large numbers.[1]   The worm may be readily shown to be present by
finding the ova in the stools.   A satisfactory treatment by drugs is
not yet known, but there is consolation in the reflection that the
parasite has probably a short duration of life, and that we may prevent
further infection by avoiding uncooked foods and drinking pure
water.

[1] When Röderer and Wagler, about a century ago, rediscovered this worm, Morgagni's
prior observation having been forgotten, they supposed that it produced the typhoid
fever then prevailing at Göttingen.   It is not difficult to see how such an error arose,
the worms having been found in the bodies of most of the victims of the fever, and
nearly coinciding in seat with the local manifestations of the disease.   In connexion
with this, it is noteworthy that the more modern theory of the etiology of typhoid fever
receives an indirect support from the fact that every person who is shown to be infested
with those very common Entozoa *Oxyuris vermicularis* or *Trichocephalus dispar* is
thereby demonstrated to have swallowed minute portions of his own or another person's
fæces.

§ II.—Diseases of the Digestive System (*continued*).

E.—Diseases of the Peritoneum.

1. Peritonitis.
2. Tubercle of the Peritoneum.
3. Carcinoma of the Peritoneum.
4. Affections of the Abdominal Lymphatic
   Glands.
5. Ascites.

PERITONITIS.

BY JOHN RICHARD WARDELL, M.D., F.R.C.P.

DEFINITION.—An inflammation of the serous membrane which invests the abdominal organs and lines the abdominal cavity. It may be partial or limited, or it may be diffused over the entire inner surface of the peritoneal sac. Effusion is almost the invariable consequence, and examination after death discovers serum, albuminous exudation, sero-purulent, purulent, or sero-sanguineous fluid and organized adhesions.

PRELIMINARY OBSERVATIONS.—Inflammation of the peritoneum is characterised by the kind of phenomena which are exemplified in the inflammation of the other serous membranes. It may occur at all ages, in every description of temperament, and under the most varied conditions of the system. It attacks the earliest infancy as well as the adult and those in advanced life, and both sexes are equally liable to the affection. It happens to the robust and plethoric, the cachectic and attenuated, and also to those whose constitution has been undermined and broken down; and whenever its distinguishing symptoms are really proclaimed it is one of the most formidable maladies with which the physician has to deal. It may come on suddenly with apparent and easily recognised symptoms, or it may supervene slowly and insidiously, and continue for a time without being detected. It may be primary when it is difficult or absolutely incapable of connexion with any foregoing or coëtaneous disease. It may be consecutive upon, or symptomatic of, some other morbid condition. It may present the sthenic or asthenic form. And it may be met with only in sporadic cases, or prevail as an epidemic. Every example of the complaint will, if carefully studied, exhibit some peculiarity—some cognizable difference in its physiognomy, if such term may be employed—dependent upon the degree of mal-nutrition, or the metamorphosis of the tissues, upon the operation of external agencies, the time of life, the amount of vital power, and the idiosyncrasies of the patient. It will be modified by the state of the depurative organs, and especially by that of the kidneys, because those deleterious and effete matters which ought to be carried off by the renal organs, when retained in the circulation, are particularly prone to institute the inflammatory process in serous membranes. When the disease is regarded in all its phases and its cardinal signs are duly observed, it exhibits a train of

phenomena peculiar to its own morbid action; and if Peritonitis, like pneumonia and certain other diseases, which formerly had always accorded to them an essentiality, is not to be deemed an essential complaint—a nosological entity, as some modern pathologists maintain —it certainly from its importance demands a distinct place in a comprehensive work like that of "The System of Medicine."

The older authors did not distinguish the inflammation of this membrane as apart and disconnected, but only as associated and confounded with the inflamed condition of subjacent organs and tissues, nor was it until the close of the last century that this distinction was made. Since that time the researches of Broussais, Bichat, Barron, Hodgkin, and more recently of Habershon, have extended our information, and given much precision to our knowledge on the subject. Sauvages remarks :—" *Enteritis mesenterica* (Peritonitis) *difficillime distinguitur ab enteritide, quacum etiam sæpe complicatur.*"[1] Cullen says it is difficult to say by what symptoms it can be recognised, and more recent authors have expressed themselves in similar language; but, as will hereafter be shown, it unquestionably displays features by which it can be diagnosticated. John Hunter thus delivers himself on this subject :—" If the peritoneum which lines the cavity of the abdomen inflames, its inflammation does not affect the parietes of the abdomen; or if the peritoneum covering any of the viscera is inflamed, it does not affect the viscera. Thus the peritoneum shall be universally inflamed, as in puerperal fever, yet the parietes of the abdomen and the proper coats of the intestines shall not be affected."[2] That these propositions are sometimes verified it cannot be denied, but according to my own experience in Peritonitis which has existed for a time, it well-nigh always happens that some of the organs and structures which it covers reveal the inflammatory products. Dr. Habershon, in a valuable article[3] on the etiology and treatment of Peritonitis, speaks with much boldness and decision on this question, and he bases his conclusions on the trustworthy ground of accumulated facts. "In 3,752 inspections recorded at Guy's Hospital," says this physician, "during twenty-five years 500 instances of Peritonitis occur, but we cannot find a single case thoroughly detailed where the disease could be correctly regarded as existing solely in the peritoneal serous membrane." He then divides them, firstly, into Peritonitis by extension from diseased viscera or direct injury; secondly, into those connected with blood changes, as in albuminuria, pyæmia, puerperal fever, and erysipelas; and thirdly, into those caused by nutritive changes, as in struma and cancer. This author then contemplates the affection, so-called Peritonitis, as nothing more than the local evidence of antecedent morbid changes pervading the whole system. Dr. Sieveking says it is the climax of nutritive derangements, certainly not to be sought for primarily, in the serous investment of the intestines.[4] The former

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[1] Classis iii. gen. xv. sp. iv.   [2] On the Blood, p. 244.
[3] Medico-Chirurgical Review, No. xliii.
[4] Croonian Lectures, *British Medical Journal*, April 14, 1866.

of these authorities denies that it is *ever* idiopathic, but he would almost seem to discard that term from pathological phraseology, as he conceives it can hardly with correctness be applied to any disease spontaneously instituted within the organism, and not dependent upon external noxious agencies or parasites.

Sometimes the lesion is but partial, in other instances it extends over the entire membrane, and doubtless it is at the outset only that it is limited, and that its diffusion gradually supervenes. Its closest analogies are pleuritis and pericarditis, and like these affections it is broadly distinguished by its tendency to effusion, adhesions by coagulable lymph, or the deposition of purulent or sero-purulent fluid. The pathologic conditions consequent upon Peritonitis, as of the other maladies now instanced, are sometimes inceptive of further disease, or they may be defensive against worst results ;[1] they may eventuate in the union or binding down of organs and parts whereby their functions are seriously or even fatally interfered with ; or this same tendency to albuminous exudation may, as in some instances of perforation, be conservative of life, the plastic deposit being the means whereby nature essays to effect reparation. But these and kindred considerations will be more fully considered when I speak of the pathology and morbid anatomy of the disease.

ETIOLOGY.—The causes of Peritonitis are often traceable to wet and cold, damp feet, damp beds, chill winds, sudden alternations of temperature, as when, after being in a heated atmosphere, the body is rapidly cooled, or to excessive fatigue—in fact to such general influences as are concerned in the production of inflammation in other viscera. It may, in a more direct manner, be induced—in a mechanical way—by invagination, strangulated hernia, surgical operations (as in paracentesis abdominis, and ovariotomy); by contusions, bruises, the wounds of cutting or blunt instruments; by displacement of some of the internal organs, or some unusual stretching or laceration of the membrane ;—by the extrusion of certain matters into the serous sac, as in hepatic or splenic abscess, rupture of the stomach, bile-ducts, spleen, uterus, urinary bladder, ureters, the ovary or some part of the sub-diaphragmatic digestive tube. It may follow or be associated with the acute disease of some organ by contiguity of structure, as in gastritis, hepatitis, splenitis, in dysentery, or in typhoid fever when the lower third of the ileum or the vermiform appendix is ulcerated. Sometimes tumours, extra-uterine conceptions, or malignant growths by the induction of pressure, or ulcerative absorption, give rise to it. The abrupt suppression of habitual discharges, more especially of the catamenia and lochia, and the sudden retrocession of cutaneous eruptions, have been enumerated; and contamination of the blood itself, resulting from the altered and imperfect action of certain of the excreting organs, enters, there are good grounds for believing, far more frequently and far more importantly as an element in the causation

[1] Sir Thomas Watson.

than has hitherto been supposed. Indeed, many attacks which we regard as idiopathic are dependent upon a common cause in the organism, but this membrane may sometimes have a greater proclivity to the condition of inflammation than any other part. Sometimes Peritonitis is metastatic of rheumatism, erysipelas, and the exanthematous fevers. Broussais repeatedly knew it succeed intermittent fever, and it is occasionally connected with fevers of a malignant type.

SYMPTOMATOLOGY.—The invasion is often sudden, but the attack may come on slowly and covertly. In the acute sthenic form there are generally rigors, followed by heat and flushings, a feeling of lassitude, aching of the limbs, head, or back, a sense of constriction and uneasiness at the epigastrium, thirst, nausea, and acute pains at some, especially the lower, part of the belly. Pressure on the abdomen, coughing, sneezing, the evacuation of the bladder or bowels, or even the erect position augments the pain; indeed whatever produces weight upon or stretches the membrane of necessity aggravates the suffering. The pain is at first localized, but it soon becomes diffused over the entire abdomen, and is a prominent sign. As the disease progresses, the pulse becomes quick, hard, sharp, and tense, and rises from 120 to 130 in the minute. In some exceptional cases it does not ascend to more than 80 or 90, and is of tolerably full volume; but as the rule it is firm, small, and cordy. The pulse is not always, however, a sure guide, as most serious attacks may be progressing under all conditions of the arterial circulation; and even pain on pressure, the most trustworthy of all individual symptoms, is not invariably to be relied upon, because it is not uniformly commensurate with the amount of lesion which really obtains. The tongue is mostly moist and covered with a whitish creamy mucus, but occasionally it is dry. The bowels have a tendency to be confined, and the urine is scanty and high-coloured. The skin is hot and dry at the earlier period of the disease, and becomes cool and bedewed with a clammy sweat before dissolution. The patient lies in the supine posture with knees drawn up, and cannot turn on either side without increase of pain. He will say that he experiences a feeling of heat, pricking, cutting, or soreness in his inside; involuntarily he relaxes the abdominal muscles, and sometimes fomentations, and even the weight of the bed-clothes, cannot be borne. The breathing becomes quick, shallow, and almost entirely thoracic, and instead of being 18 or 20 it may be 50 or even 60 in the minute. The downward pressure of the diaphragm is instinctively as much as possible avoided, because it moves the abdominal organs, and all movement gives pain. The passage of flatus along the bowels is followed by the same effect. With regard to the pain, which is a cardinal sign, it presents some differences; sometimes it is permanent, in other cases it is paroxysmal, assuming a spasmodic character, and in a few rare examples it is not present in marked degree. As the rule, it is the chief and most reliable symptom.

There is always between this disease and the features great sympathy. The face becomes pale, the cheeks collapse, and the eyes seem set and sunken in their foramina. It assumes the Facies Hippocratica, or what the French term the *Facies Grippée*. Nausea and vomiting often come on with the other symptoms, the ejected matters being a mucoid, biliary fluid; or, in the case of obstructed bowels, the vomited matters may be stercoraceous. Tympanitis is never absent, and often very distressing. The loss of tone in the muscular coat, and the irritation which is conferred on the mucous surface of the alimentary canal, account for such condition. The distension varies in degree. In those whose bodies are flabby and resistless it is often excessive, whilst in the robust and muscular it is in less amount. If the diaphragmatic covering becomes inflamed, singultus often occurs; when the serous coat of the stomach is involved, sickness is urgent; if that of the urinary bladder, there is strangury; and the inflamed tunic of the kidneys will produce ischuria renalis. Percussion elicits the loud tympanitic note, especially in the umbilical and epigastric regions. When there is effusion of serum—which, of course, gravitates to the lower parts—the line of dulness can be most distinctly observed, and it is in some measure altered according to the position of the body. Palpation can only be had recourse to with great care, as the extreme tenderness and muscular resistance prevent much manual examination. When effusion has taken place, and coagulable lymph has matted the intestines together and formed roughened deposits on the liver, spleen, or some tumour, and when albuminous concretions adhere to the parietal peritoneum, the flat hand laid on the abdomen feels a peculiar thrill or vibration, which is most distinct during inspiration. This sign only obtains when the lymph is thrown out on a resisting basis. Auscultation discovers a creaking friction sound, which is variable in character and intensity, and can only be present for a short time, as of course, on the advent of adhesion, nothing can be heard. The physical signs of pericarditis and pleuritis are far more common. Death is ushered in by quick and thready pulse, cold and clammy surface, loss of heat in the feet and legs, accelerated and laboured breathing and general declension of power, the mind being often clear and collected to the last. Pemberton says the patient frequently expires on the sixth, seventh, or eighth day. But it is equally true that the fatal issue often occurs in two or three days. In puerperal Peritonitis the average duration of the disease has been shown to be about thirty hours, and sometimes, as in perforation, it may be even less than ten hours. When the affection assumes a more chronic form, the patient may live so long as thirty or forty days.

The asthenic type of Peritonitis occurs in the cachectic, and those whose vital powers have been undermined by some previous disease. It is that form which is seen as metastatic of erysipelas and rheumatism, and in connexion with the exanthems, malignant fevers, puerperal women, and when there is perforation of some part of the digestive tube. It proclaims contamination of the blood and want of

vital power. The effusion is sudden, large in quantity, of debased character, and notably deficient in organizable plasma. The pulse is soft and feeble, the surface soon becomes moist, and all the phenomena proclaim declension of vitality.

When the disease terminates by *resolution,* a gradual improvement of all the symptoms becomes observable. The symptomatic fever declines, the pain is less urgent, and pressure can be borne on the abdomen; the skin is moderately moist, but not below the ordinary temperature; the tongue looks cleaner; the pulse is slower, fuller, and softer; the respiration is more normal, being less frequent and not so thoracic; the alvine evacuations are freer and more natural; and there is generally a copious secretion of urine, which contains an abundance of lateritious deposits. Sometimes moderate diarrhœa or diaphoresis are critical discharges. The sickness and vomiting cease, the tympanitis and feeling of distension obtain in less degree, and the patient can extend his legs and lie on either side with more freedom and ease. Lastly, the countenance, which had hitherto been so faithful an index of the complaint, looks calmer and more natural, it having lost much of the sunken, collapsed appearance above described.

One of the most frequent results is *effusion;* indeed, the affection cannot assume a well-marked and typical character without one or other of the inflammatory products being thrown out, and these, as to their proportion and quality, are varied in every individual example. In the earlier stage of the attack the effusion is but small, and not such as in marked manner to increase the size of the abdomen. It gravitates into the pelvis and the iliac fossæ. It can be detected by percussion over the lower parts of the belly, and there are general signs which indicate its presence. When it increases, the pain becomes a less prominent symptom, the pulse is softer, there is a feeling of weight and dragging in the body, chilliness and a diminution of animal heat, the extremities having a tendency to become cool. In such cases as are metastatic of some other complaint, the effusion is much more rapidly generated and the serous proportion is relatively very large. Andral records an illustration which was metastatic of rheumatism, and which ran through its course to a fatal termination in three days, and the autopsy showed an enormous quantity of serum tinted with the colouring matter of the blood, and some floating flocculi and false membranes. When pus is secreted, rigors are a common symptom, with febrile exacerbation in the evening, and the pulse is quicker. It is not, if in any notable quantity, absorbed. It finds an exit either by forming an ulcerated opening into the bowel, which is always fatal, or, which is much more common, it establishes a fistulous passage by way of the psoas muscle, or through some part of the abdominal walls. In this tendency to appear at the surface it seems to obey the law of an ordinary abscess.

Inflammation of the peritoneum rarely ends in *gangrene,* and it is still more rare for any considerable portion of the membrane to become gangrenous. When it has come on, it has generally been at or about

the vermiform appendix, or when some part of the bowel has been unduly stretched or strangulated; and, according to Abercrombie, it is invariably accompanied with false membranes. The sudden cessation of pain, singultus, coldness of the surface, thready compressible pulse, general declension of strength, and the Hippocratic countenance, are indicative of this condition.

Sometimes the acute gradually passes into the chronic form, when, as before remarked, the patient does not die until after five or six weeks. He may live even several months. In such cases the effusion may not be absorbed nor yet evacuated, or a fistulous communication may have been produced, and all the conditions of asthenia usher in the mortal event. Again, in other examples, the serous fluid will be absorbed, the adhesions become firm and organized, or the sero-purulent or purulent matter be discharged, and slow recovery result.

The phases which the inflammation of this membrane may assume are very varied; and it is only by the study of a large number of examples that the physician can anticipate and comprehend the modes of its progress. Sometimes that cardinal symptom pain, upon which such emphasis has been laid, only obtains at the outset; and notwithstanding its subsidence, the malady goes on. Occasionally, as in pleuritis, there may be little or no pain from first to last, whilst rigors and hectic and wasting pronounce still the seriousness of the case at a time long after that period when danger is generally thought to have passed away, and a large collection of pus is contained in the cavity; or the acute symptoms may rapidly subside under a properly directed antiphlogistic treatment, and the condition of simple ascites will only appear to be present; again, disease instituted in some of the abdominal organs will greatly modify the affection after it has become chronic. In this state adhesions alter the configuration of the abdomen by large masses of fibrin being deposited together, by the soldering of the intestinal convolutions, the agglomeration of one organ to another, or by the formation of separate collections of matter in distinct septa resembling independent abscesses. It sometimes happens, too, that the belly becomes soft and flabby, and, instead of improvement succeeding this disappearance of the tension, convalescence is slow and protracted. From what has now been said, it is obvious that the chronic condition is far from being uniform in its phenomena, and that the pathological changes may be diverse and multiform.

VARIETIES.—Broussais and some other authorities speak of the induction of Peritonitis by the exudation of blood into the abdominal cavity without solution of continuity in any of the blood-vessels. I have never seen such an instance, and these examples must be extremely rare. Such sparse exceptions are to be associated with the hæmorrhagic diathesis, the predisposing causes being the sanguine temperament and a marked tendency to inflammatory complaints. According to Broussais, the pulse is at first full, but soon becomes soft and compressible, the pain very acute, often intermittent, and coldness

of the extremities and convulsions quickly close the scene.[1] Laennec was one of the first to draw attention to hæmorrhagic exudations of serous membranes, and Rokitansky attributes such tendency to the tubercular cachexia, the diseased condition of the blood resulting from cirrhosis of the liver, the scorbutic constitution, and the dyscrasia of drunkards.   The effect of specific poisons, such as induce the various febrile diseases, and that anomalous condition of the blood now spoken of in which its fibrinous constituent is diminished, and its serous part augmented, are to be enumerated in the causation of this hæmorrhagic exudation.   When the blood having this origin is discovered in the peritoneal sac, it is in large quantity, very red, and in varying proportions mixed with serum.

There is another description of Peritonitis which systematic writers have recorded, and to which the name of *latency* has been given. It has been said to attack those labouring under some other ailment, the feeble and attenuated, the aged, the insane, and such as exhibit a low degree of vitality.   Its symptoms at the outset are masked and difficult of recognition, and, when recognised of the asthenic type, the features present those distinguishing traits before insisted upon as being characteristic of this complaint.   It is evident that such examples are nothing more nor less than secondary affections like unto pneumonia in albuminuria, pleuro-pneumonia when intercurrent in phthisis, pericarditis in rheumatism, and arachnitis in continued fever.

Non-plastic or Erysipelatous Peritonitis.—This is seen as the sequel or complication of the exanthems, in adynamic fevers, and in puerperal Peritonitis.   Its essential condition is some hæmic change, and it is characterised by asthenia.   It is met with in worn-out and undermined constitutions, in the unhealthy, and in those who have had some other malady.   Its supervention is sudden, and it runs its course with great celerity.   It does not bear an antiphlogistic or lowering treatment, and is only benefited by stimulating and sustaining remedies. According to Abercrombie, "the symptoms are sometimes slight and insidious, but sometimes very severe; and they are chiefly distinguished by the rapidity with which they run their course, and by a remarkable sinking of the vital powers which occurs from an early period, and often prevents the adoption of any active treatment. A remarkable circumstance in the history of this affection is its connexion with erysipelas, or with other diseases of an erysipelatous character."[2] Illustrative of this form of the complaint he gives the instance of a woman who had erysipelatous inflammation of the throat, who was very suddenly seized with abdominal pain and vomiting, and who gradually sank in forty-eight hours.   The necroscopy discovered a large quantity of pus in the peritoneal sac.   And he gives other and similar examples.   This physician also refers to an epidemic of erysipelatous character which occurred amongst the children in the Merchants' Hospital, Edinburgh, in 1824.   The disease was of mild

[1] Broussais, Histoire des Phlegmasies ou Inflammations chroniques.
[2] Pathological Researches on the Diseases of the Abdominal Viscera, 3d edit. p. 181.

type. In all the cases there was throat affection, consisting of a raw, red appearance, swelling, and aphthous crusts. Two of the little patients speedily sank, and inspection revealed pus in the abdominal cavity. Abercrombie draws a comparison between this epidemic and one of diphtherite, as it was then named, which appeared two years afterwards, and he believed them to be congeners. The correctness of this opinion later years have confirmed. Between diphtheria and erysipelas there is great resemblance. They are both referrible to general blood change, and, as it has been well remarked, are associated with a large group of maladies which stand in close relation with pyæmia.[1] The kind of Peritonitis spoken of occurs with a depressed vitalism, consequent upon toxæmic agents imbibed from without or formed within the organism by its own power of genesis; and the term non-plastic well applies to the ostensible difference which there is between this type, deficient in organizable plasma, and the adhesive form of inflammation.

Perforation of the Peritoneal Membrane.—There is no form of Peritonitis which is so fearful and fatal as that in which there has been positive solution of continuity of the membrane, because this accident generally implies the extrusion of some secretion or fluid or substance into the serous cavity. Several of the older authors mention this occurrence, and some vaguely attribute such openings to worms—a *possibility*, as we know from Andral's case, in which lumbrici passed into the cavity; but this event is exceedingly rare. There is no doubt that in nearly all these recorded instances the real cause of such perforations was ulcerative destruction, or cadaveric change, which former writers had not recognised with that facility and certitude which distinguish the acquisition of modern pathologists. Perforation may be produced in a great variety of ways, by penetrating wounds made by sharp or blunt instruments, the crushing effect of accidents, lacerating the solid or hollow viscera, or the parietal peritoneum; corrosive poisons, the giving way of the uterine walls during parturition, the softening of a fibrous tumour attached to the uterus and the contents being extravasated; the bursting of a Graafian vesicle, of a mesenteric gland, of a tubercular deposit, of the urinary or gall-bladder; from calculi, from the evacuation of some collection of purulent matter, as in empyema; burrowing through the diaphragm, in abscess, as before remarked, of the liver, spleen, or kidney, in pelvic abscess, and from other causes. Mr. Hulke lately recorded an instance of renal abscess bursting into the peritoneal sac, which occurred in an unhealthy-looking maid-servant who was admitted into Middlesex Hospital for hip disease, and which ended fatally. The inspection discovered puriform serum in the peritoneal cavity, and the peritoneal surfaces were coated with a soft yellow lymph. The right kidney was a mere sacculated pouch, and it was ruptured at its upper end.[2] The more common cause of perforation is ulceration, commencing in the mucous mem-

[1] Dr. Russell Reynolds, art. Erysipelas, vol. i.
[2] *Lancet,* Jan. 23, 1866.

brane, of some portion of the digestive tube, and penetrating through the muscular and serous coats. It may be referrible to softening of the intestinal wall (*ramollissement gélatiniforme*), or to cancerous disease, especially when the cancerous deposit encroaches upon, or absolutely blocks up, the passage. When the accident is from this cause, it is mostly observed in the stomach, colon, or cæcum.

The symptoms are sudden, often violent. Frequently the patient at once falls into collapse. Andral says, that sudden increase of prostration and rapid change of the features are sometimes the only symptoms denoting the accident of perforation. Sometimes there is febrile excitement, as evinced by increased heat of surface, hard pulse, and urgent thirst. In the great majority of cases remedies seem inoperative; the disease rapidly becomes diffused over the surface of the sac; whilst vomiting, dorsal decubitus, quick and feeble pulse, loss of animal heat, and sunken and collapsed features, too truly indicate the powerful impress which has been made upon the circulatory and nervous systems, the mental faculties, generally, remaining unaffected to the last. In those very exceptional cases in which recovery does take place the vomiting begins to subside, the distension to decline; the pulse becomes softer, fuller, and slower; the face is less haggard, the patient sleeps more tranquilly, and the temperature of the body is more natural.

When the stomach is the seat of perforation, as it sometimes is, by simple or specific ulcer, the phenomena are precisely those which obtain when any other part of the sub-diaphragmatic tube gives way. Ulceration of this organ is most frequent in females. Dr. Brinton found that in 654 cases 440 were in females, and 214 in males. He also says that in the former sex one-half occurred between the ages of 14 and 20.[1] It happens to children. Dr. Lee knew perforation of the stomach of a girl of 8, and in that of a boy of nine years of age. The opening is most frequent at the splenic end, and that part is also most prone to gelatiniform softening. It may give rise to hæmorrhage. Habershon gives an example in which the splenic and pancreatic arteries were opened. It does not absolutely follow that death shall always eventuate, because adhesion may take place between the point of ulceration and the abdominal walls, or one of the solid viscera, or a communication may be established between the stomach and the colon, or the duodenum, or a gastric fistula may be formed externally, or through the diaphragm into the thorax. The last two named are very uncommon, but possible contingencies. Abercrombie gives an example

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[1] Dr. Brinton gives the following relative proportions per cent. of the locality of perforations which ended fatally by Peritonitis:—

Posterior Surface	2
Pyloric Sac	10
Middle	13
Lesser Curvature	18
Anterior and Posterior Surface at once	28
Cardiac Extremity	40
Anterior Surface	85

of the kind of Peritonitis now considered. A young woman had been affected with dyspeptic symptoms and epigastric pain for some months. On Nov. 26th, 1824, she was heard to scream violently, and when approached was unable to express her feelings except by violently pressing her hand against the pit of the stomach. The abdomen became tender and distended, and she continued in extreme suffering till the 27th, when she died twenty-nine hours after the attack. On the inspection of the body the cavity of the peritoneum was distended with air, and likewise contained upwards of eight pounds of fluid of whitish colour and foetid smell. There was slight but extensive inflammatory deposition on the surface of the intestines, producing adhesion to each other, and to the parietes of the abdomen. In the small curvature of the stomach was a perforation which admitted the point of the little finger.[1] This author gives another case in the person of an elderly gentleman, who was suddenly seized with excruciating pain at the stomach, accompanied by vomiting, coldness, and quick pulse. The abdomen became tense and tender, and he sank in thirty hours. Necroscopy exhibited near to the pyloric opening an ulcerated hole larger than a shilling, to which the liver formed a base, and a little below a perforation of the calibre of a quill through which the contents of the stomach had escaped and caused fatal Peritonitis.

The duodenum is less liable to this accident than the stomach; but its serous tunic does sometimes give way under the ulcerative process. Mr. Curling was the first to observe that the glands of Brunner are apt to pass into ulceration during the progress of severe burns, and from this cause Peritonitis may in a secondary manner result. In twenty-two autopsies made by Louis in enteric fever, in only two cases was the villous surface of the duodenum found ulcerated. In fifteen examples of that disease examined by Jenner, and in twenty by Murchison, no morbid condition was detected in this organ. Its ulceration in all its characteristics and consequences very nearly resembles that described of the stomach. Habershon says several cases have come under his observation, the early symptoms of the ulceration being slight until fatal Peritonitis had been set up by perforation. In other instances violent vomiting produced the accident. Hodgkin relates the instance of a young woman aged twenty-four, who was admitted into Guy's with urgent vomiting, small and feeble pulse, and who shortly after died of fatal Peritonitis caused by a small ulcer in the duodenum. Habershon gives an interesting example in a young woman, aged eighteen, admitted into Guy's February 19th, and who died October 4th, 1860. At first the prominent symptom was vomiting; after a time diarrhœa came on, and the emaciation increased. Examination of the body showed behind the first portion of the duodenum and close to the pancreas a collection of offensive pus, and a perforation a quarter of an inch in diameter was discovered. From the histories of six cases recorded by Dr. Andrew Clark,[2] he concludes

[1] Abercrombie's Diseases of Stomach, 3d edit. p. 34.
[2] *British Medical Journal,* June 22, 1867.

that the event is sudden, after food, and that the pain never leaves its place of origin. In the examples given by this physician there was no sensation of something having given way, nor of heat diffusing itself over the belly. This organ is more frequently perforated by secondary than primary disease. The malignancy of neighbouring viscera is sometimes extended to its parietes, as in cancer of the stomach, liver, spleen, pancreas and lymphatic glands, and its consequent rupture is followed by Peritonitis, which ends fatally.

With regard to the jejunum it is rarely found morbid, and assuredly no part of the digestive tube possesses such an immunity from disease. I have known no instance of its perforation. Neumann and Hufeland, however, have recorded an example of this event.

Perforation more frequently occurs in the lower third of the ileum, and near to the ileo-cæcal valve than in any other part of the intestines. Of ten cases by Louis, it was within a foot of the valve. Of ten cases given by Stokes, in nine it was within twelve inches of the valve, and one was in the cæcum. Of eleven by Murchison, nine were within twelve, and two within eighteen inches of the same place. Bartlett saw it forty-four, and Bristowe seventy-two inches from the same place. The parts next in order of prevalence are the cæcum and vermiform appendix. Louis was one of the earliest observers of the facts now noticed. It has long been broadly and familiarly known that the agminate glands which are proper to the ileum, and the solitary glands which are scattered throughout the villous coat of the digestive tube, are in enteric fever very prone to take on the ulcerative condition, more especially the patches of Peyer, and occasionally it happens that after the mucous and muscular coats have been destroyed the peritoneum gives way. These glands are not in like manner predisposed to disease in the course of any other acute affection. The vermiform appendix has in repetition been found the seat of fatal Peritonitis, not only in enteric fever, when sometimes only a very minute orifice can be discovered, but from the impaction of some foreign body, as the seed of fruit, a kernel, a piece of bone, a piece of indurated fæcal matter, or even the single bristle of a tooth-brush. Of eight cases of perforation given by Louis, seven were in the young and vigorous, and it may here be observed that more recent writers, as Jenner, Murchison, and Bristowe, have shown that it chiefly occurs between the ages of fifteen and twenty. Of the eight cases by Louis, with a single exception, the disease commenced with continued fever, nor did the febrile phenomena assume any severity of character until the advent of the perforation. In four there had been diarrhœa, but only in one were the bowels much harassed. Tweedie says the state of the bowels, either as to the presence or absence of diarrhœa, is not to be depended upon, as it sometimes happens that the evacuations are healthy when the bowel gives way. Three were quite convalescent when the opening occurred, and a fourth appeared to have fully recovered from an attack or enteritis.

Since Louis wrote his account much information has been accumulated on this particular subject. It is now well known to all who have made the various forms of fever a special study, that there is no precise correlation between the gravity of febrile symptoms and the occurrence of perforation. The diarrhœa may have been a distressing and persistent symptom, and yet the points of ulceration may not have been either numerous or deep; on the other hand, in cases regarded as mild forms of fever the bowel may very unexpectedly burst, and this event is generally at a later date of the attack, or during convalescence. Tweedie has known it take place when the patient has so far recovered as to leave the house. Dr. Murchison lately published an apt illustration.[1] Some time ago I had under my care a girl in enteric fever who became quite convalescent, and at the end of six weeks, after eating a hearty meal of solid food, Peritonitis supervened, and she died in twenty-two hours. Peacock saw it come on so soon as the eighth, and Murchison on the ninth day of fever. Louis noticed it so late as the forty-second, and Jenner on the forty-sixth day. Of thirty-two cases given by Murchison, perforation occurred during the second week in eight cases; during the third week in six, during the fourth week in nine, and after the fourth week in nine.[2] Louis says if in acute disease, and in an unexpected manner, a violent pain in the abdomen supervenes: if *this pain* is exasperated by pressure accompanied by rapid alteration of the features, and more or less promptly followed by nausea and vomiting, we may believe and announce that there is perforation of the intestine.[3] Pain is not a symptom in all cases continuous up to death. It sometimes notably abates, and in exceptional examples ceases entirely for several hours before dissolution. Jenner saw a patient in whom there was no pain at all, vomiting and cold extremities being the only symptoms. Tweedie asserts that the symptoms of this event are not uniformly well pronounced. The accident may be masked by delirium so considerably that the time of perforation and its absolute occurrence may be uncertain.

Dr. Stokes gives particulars relative to nine cases which occurred under his own observation.[4] These happened during fever; one in catarrhal fever, two after acute enteritis, and in one case hypercatharsis produced by an overdose of salts was the cause. In several of these nine instances there had been diarrhœa. He also comments upon a fact worthy of notice, that in three were produced irritation of the bladder and inability to pass urine. In all, inspection revealed ulceration of the muciparous glands; and respecting the time which the patient lived after the initiatory symptoms of perforation, it varied from twelve to one hundred and twenty hours. Stokes also says that the average duration, deduced from nineteen cases which he had collected from various sources, was twenty-nine hours. Louis' patients lived from twenty to fifty-four hours. Murchison has known death

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[1] *British Medical Journal*, Dec. 2, 1865.
[2] On Fever, p. 508.
[3] Recherches Anatomico-Pathologiques.
[4] Cyclop. Pract. Med.

follow in four hours, and not until one hundred and five hours. I have known it from seven to twenty-three hours. The period subsequent to the accident must needs be influenced by a variety of circumstances, such as the character of the antecedent or coetaneous disease, the vital powers of the patient, the extent of the orifice, and the kind and quantity of lymph thrown out, the part of the bowel, and the conditions favouring or opposing adhesion. If in a fever of the adynamic type, when the powers of the system are much reduced, the shock may be such as at once to usher in a fatal collapse. If the opening be in immediate apposition with another coil of the bowel, a solid organ, or the walls of the abdomen, the extrusion of the contents of the canal may for a time be arrested. Bristowe relates a case in which the patient lived fourteen days after perforation. I remember an instance in enteric fever in which there was a hole that would have admitted a swan-shot on the lower part of the ileum, but depositions of pearly lymph had so effectually sealed up the opening that none of the intestinal contents had escaped. When, however, they do escape, the inflammation becomes so intense that remedies are generally powerless. Chomel, Louis, Rokitansky, and Jenner say it is *always* fatal. Tweedie, Todd, Ballard, Fox, Bell, and Murchison aver that they have known recovery. The last-named relates the instance of a girl of sixteen, who, on the thirty-first day of fever, was suddenly seized with severe pain and tension of the abdomen, urgent vomiting, and all the symptoms of collapse. A grain of opium was given every second hour, and during the first thirty-six hours ten grains were taken. The patient made a tedious recovery, and was discharged from the hospital fifty-five days after the commencement of the Peritonitis.

In some exceptional examples, the more formidable symptoms will apparently subside, and life be preserved for even several days. This deceptive kind of amendment should not, however, throw the physician off his guard; he should not forget those grave and alarming indications which pronounced the existence of the accident, as it almost invariably proves that the mortal end has only been deferred, not averted. In the case observed by myself, if there was no absolute escape of the intestinal contents, the soft lymphic plug could not for any great length of time have sufficed to act as a barrier to extravasation. Some slight strain, as in the evacuation of the bowels, coughing, sneezing, or the mere motion of the body, might doubtless have been sufficient to remove the non-organized albuminous deposit, and render the opening free. Notwithstanding the well-nigh hopelessness of all cases in which there is positive solution of continuity, it is from pathological reasoning a possibility that recovery may succeed. Nature attempts to repair the lesion by throwing out plastic materials, and if these,—by utter rest, and by opiates subduing the peristaltic action of the bowels,—be allowed to lie in contact with the breach sufficiently long to become permeated with new vessels—to be organized—the orifice may be repaired

ation, however, can only be effected when the hole is small,
t is but a mere possibility.

the first symptoms of perforation are nearly always distinct
.e, in exceptional cases they may be ill-defined and obscure;
ray gradually assume increased severity. They will be
by the size of the aperture; for instance, the solution of
, when it takes place in the appendix, is sometimes very
id the escape of irritant matters inconsiderable. The orifice
st be small and by degrees enlarge, and relatively with the
nt (and consequent greater extravasation of liquid and fæcal
will increase the irritation conferred to the sac and the more
phenomena of inflammation. Confirmative of these asser-
John Harley may be cited. "In some cases," says this
"the perforation has taken place so gradually, the aperture
so small, and the extravasation so inconsiderable, that the
of Peritonitis come on and attain their maximum very
and without any sudden increase in the severity of the
."[1]

on is occasionally perforated in fever, but it is much less
this result than the parts last named. Chomel, Brinton,
d Murchison mention five instances. In two out of these
opening was at the junction of the transverse and descending
l in the three others at the junction of the sigmoid flexure
rectum.[2] The last-named authority lately gave a good
f the giving way of the large intestine. "A young man of
was admitted into the Fever Hospital, Aug. 23, 1865; he
ill fourteen days, and on admission was very ill of typhoid
l Peritonitis. The pulse was quick and feeble, the body
y distended and tender, the motions frequent and watery,
breathing thoracic. He died Sept. 7. Inspection discovered
surface of the peritoneum to be coated with a thin layer of
lich could be stripped off with a knife. There were three
as in the large intestine, one about three and a half inches
: valve, and two in the sigmoid flexure. There were no
f the bowel in the serous sac."[3]

espect to the average of perforation in fever, Murchison
t out of 435 autopsies recorded by Bretonneau, Chomel,
Forget, Waters, Jenner, Bristowe, and those made at the
'ever Hospital, it occurred in 60 cases, or in 13·8 per cent.[4]
ly happens in about three per cent. of those who have enteric
more frequently amongst males than females.

nic dysentery, sometimes, after ulceration has destroyed the
nd muscular coats, the peritoneum is penetrated. In such
the special and general symptoms, which characterise the
isease, point to a correct diagnosis. In cancer of the bowels
n may occur: it is more frequent in the large than small

n of Medicine, vol. i. p. 570. *   [2] Murchison on Fever, p. 551.
A Medical Journal, Dec. 2, 1865.   [4] On Fever, p. 511.

intestines, and Rokitansky says the colon is almost exclusiv[e]
the seat of cancerous degeneration. I saw in consultation some ti[me]
ago a gentleman labouring under diffuse Peritonitis, which h[ad]
evidently been caused by a large hard tumour, the size of a crick[et]
ball, in the left hypogastric region. The stools were flattened, but [the]
passage was evidently quite patulous. I gave it as my opinion tha[t it]
was a case of cancer of the large bowel. A surgeon was at this ju[nc]
ture called in, and he strangely enough proposed Amussat's op[era]
tion merely to give exit to the flatus, when large pieces of fæcal mat[ter]
were voided, but fortunately that suggestion was overruled by two [of]
the most eminent members of the profession. In the course of a [few]
days the patient died. Perforation was announced by a sudden [and]
terrible increase of pain, small pulse, sunken features, and cold extre[mi]
ties. The autopsy revealed abundant proofs of foregoing and pre[sent]
Peritonitis. There were several pints of serum in the abdomen, wh[ich]
contained loose flocculi; the descending colon was adherent to [the]
abdominal walls, and a little above the sigmoid flexure was a clea[n]
cut, punched hole, the size of a small pea, through which a la[rge]
quantity of thin feculent matter had passed into the peritoneal s[ac.]
The upper third of the rectum, and the opening into the sigm[oid]
flexure, were the seats of cancerous deposit, and the canal w[as]
patulous.

Habershon divides perforations into two great classes, those wh[ich]
arise from disease commencing in the intestine itself, as by the ulce[ra]
tion of fever, dysentery, cancer, and the various forms of insupera[ble]
constipation, and those in which perforation is from without, as [in]
strumous Peritonitis, ulceration of the stomach extending to the tra[ns]
verse colon, hydatids, and abscess of the liver, calculi, abscess in t[he]
other solid viscera or abdominal walls, cancer, extra-uterine fœtati[on,]
and external injuries.[1] It may be caused by laceration of the ga[ll]
bladder. Barthez and Rilliet mention a case in a girl of twelve wh[ich]
in fever, and Murchison gives another instance in a young man [of]
nineteen, who was suddenly seized with Peritonitis on the fiftee[nth]
day of the fever, and who died in twenty-six hours. It is rar[ely]
observed as the result of tubercle. Sir Thomas Watson, in [his]
large experience, only remembers a single instance. Of fifty-[six]
cases collected by Habershon, four only were from strumous d[is]
ease. Jenner once knew a softened mesenteric gland give w[ay]
during fever, and Buchanan saw a fatal case of Peritonitis fr[om]
the bursting of a softened embolic deposit in the spleen o[f a]
typhous patient.

Puerperal Peritonitis. — In the discussion of this part of [the]
subject I may here observe that it is not my purpose to enter u[pon]
the consideration of puerperal Peritonitis as it occurs epidemica[lly,]
but as I believe with many other writers that puerperal women [are]
liable to a simple form of Peritonitis, its description necessarily co[mes]
within the limit of this article. Sporadic cases from time to time o[ccur]

[1] Diseases of the Abdomen, 2d edit. p. 530.

t the diffusion of the disease, but even then it is right to observe
nost caution, as so much doubt is always involved with regard
contagious nature. Inflammation of the serous covering of
:rus and its appendages may, I believe, supervene as an inci-
circumstance, without the superaddition of a specific poison.
·eat effort of the organism, the irritable condition of the body,
ie exhaustion of expulsive endeavours, the long distension of the
and the abdominal walls, and their sudden contraction; the
i of opposed surfaces in the abdomen during labour, and the
:xcitation given to the circulatory and nervous systems, may
e Peritonitis. Other causes operate in the production of this
such as injuries inflicted during instrumental delivery, in turn-
lhesion of the placenta, the use of cold affusions in flooding,
ie improper administration of stimulants. Contamination of
)od, originating in the body itself, without reference to external
:s, as when absorption takes place from putrid coagula or a
)f retained placenta, is another mode by which the malady is
ited. In uræmic poisoning, as before remarked, the serous
:anes are predisposed to inflammation, and the blood vitiation
parturition resembles this cause.
re is, I need scarcely say, still much conflict of opinion relative
real nature of abdominal inflammation after child-birth. By
it is yet maintained that Peritonitis and puerperal fever are
:al—that these terms express but one affection. It is true that
:ge proportion of those who die of puerperal fever the peritoneum
imed, but this membrane is not *always* involved; and although
rm of inflammation accompanies this disease far more frequently
iny other form, yet puerperal fever is something still more. Of
itopsies of puerperal fever, given by Tonnelli, in 193 were traces
itonitis; in 29, or one-eighth, there were no traces whatever. Of
·es examined by Lee, the peritoneum and uterine appendages
:nflamed in 32, or in the relative proportion of 8 cases out of
11. Dr. Bartsch, in a report of the Midwifery Institution at
a, records the morbid appearances of 109 cases of those who
·f puerperal fever, and in this report puerperal fever is distin-
·l from Peritonitis and metritis. "The cases of puerperal
·he says, "occurred *seldom under the form of puerperal Peritonitis,*
·nerally as inflammation of the uterine veins, giving rise to the
:tion of pus in these vessels, and the general symptoms accom-
ig its absorption."[1] Let any one, says Fleetwood Churchill,
.re a case of simple inflammation of the womb or peritoneum in
bed with a case of epidemic puerperal fever, their symptoms,
:, and the effect of remedies, and I do not think a doubt will
i upon his mind, that although the latter is a local disease it is
clusively so.[2]
symptoms common to this form of Peritonitis may come on in a

few hours, a few days, or even so long as two or three weeks after delivery. Pains and rigors are generally the first indications, and pain on pressure is more distinctly felt at the hypogastrium than at any other part. The skin is hot, the cheeks are flushed, the pulse is quick, and the respiration hurried. The pain soon radiates from the hypogastrium into the iliac fossæ, and then to the other parts of the abdomen. It is not always severe, and is sometimes characterised by paroxysmal attacks, the patient being free from suffering during the intervals; nor can it be said that this symptom pain is pathognomonic of puerperal peritonitis, because post partum uterine pain may be urgent when there is no co-existent inflammation, and there may be inflammation with little or no abdominal pain. Churchill asserts that he has seen five or six cases of intense Peritonitis as proved by dissection, in which there was neither pain nor tenderness;[1] and Ferguson records that he has known nineteen cases in which there was no pain.

The abdomen suddenly becomes large, more quickly and to a greater extent than in any other kind of Peritonitis, which may be accounted for by the often relaxed and resistless condition of the muscular system of parturient women, and because the abdominal walls have been so recently distended. At the onset of the attack the uterus can be felt above the pelvic brim, soft, flabby, and uncontracted, but as the distension obtains in greater degree it cannot be distinguished. The lochia are at once diminished or suspended, or their absolute suppression may precede the inflammatory phenomena. If the milk has begun to flow, its secretion is arrested; if it has not begun, it is prevented. If the mammæ have been full and rounded, they fall in, and are flaccid and smaller. The pulse varies, but it is always above, in the great majority of cases greatly above, the normal standard. In non-inflammatory, uncomplicated cases the circulation may be accelerated, for a day or two, or two or three days, but there is a gradual declension of its frequency from the time of delivery. If, however, after delivery the pulse shall have fallen to, or near, its natural number, and it then suddenly begins to rise, accompanied by local pain, higher temperature, thirst and diminished secretions, the cause is often obvious.

After-pains may be confounded with those of inflammation. They come on soon after delivery, but decrease in force and frequency as time wears away. Peritonitis does not come on so soon, and its symptoms become more and more proclaimed, instead of diminishing. After-pains are associated with a firmly contracted uterus; Peritonitis with a relaxed uterus. Remedies which relieve the former are useless or harmful to the latter. In the one affection the circulation may be natural; in the other it is never so. At the first the diagnosis is very difficult, because after-pains may be followed by inflammation, and for a time the symptoms be mixed up; but the progress of the case leads to a correct conclusion. When puerperal Peritonitis is accompanied

[1] Diseases of Women, 5th edit. p. 783.

with intestinal irritation and the inflammation has extended to the mucous membrane, sickness and diarrhœa may be urgent. When the malady terminates by resolution, the pain abates, the tympanitis declines, the pulse becomes fuller and slower and softer, the skin cooler and moist, the tongue cleaner, the lochia are re-established, the breasts become rounded and milk begins to flow, the legs can with more comfort be extended, and the patient can lie on her side. The conditions of approaching dissolution are—weak and thready pulse, varying from 120 to 160; the abdomen keeps distended and tender, cold clammy sweats come on, the extremities become cold, the breathing is quick, shallow, and thoracic, she lies on her back with legs drawn up, the features are sunken, and the mind often remains calm and clear to the close.

Perityphlitis.—This particular form of disease has been more fully described by French than British pathologists. MM. Husson and Dance[1] give an excellent account of the affection; and it is also well described by Dupuytren, Menière and Duplay. Amongst the English authors may be named Copland,[2] Syme,[3] Craigie,[4] Farrall,[5] Burne,[6] Sellar,[7] and West.[8] The disease originates in the tunics of the cæcum, and by some it has been named pericæcal abscess; the glands or follicles of this organ at the first become inflamed and then pass into the ulcerative condition. The ulceration of this part of the large bowel may insidiously destroy the mucous membrane, implicate the sub-mucous cellular tissue and peritoneal coat, and either cause inflammation and lymphic adhesion of the latter, or its fatal perforation. When agglutination occurs the lesion may be arrested. Craigie defines the malady to consist in inflammation and suppuration of the cellular tissue connecting the cæcum to the quadratus lumborum muscle and other parts, or in inflammation and ulceration of the mucous membrane of the cæcum; and Sellar says its pathological seat is in the cellular tissue between the fascia of the iliacus internus and the coats of the cæcum.

The causes of perityphlitis may be referred to the peculiar position of the cæcum, as well as to other circumstances. It is attached to the muscles of the right lumbar region, and its sacculated pouch depends below the ileo-cæcal outlet, and, as all physiological anatomists observe, its contents have to be propelled against gravity; and it thus may become distended with fæcal matters, and such irritation be instituted by its distention and pressure as to set up inflammation of the lining membrane. Again, hard and indigestible articles of food, the stones of drupaceous fruits, seeds, pieces of bone, and metallic, porcellanous, and vitreous fragments have been known to give rise to it. The com-

[1] Mémoire sur quelques Engorgements inflammatoires qui se développent dans la Fosse iliaque droite; Répertoire d'Anatomie, &c. t. iv. p. 74. Paris, 1827.
[2] Med. Dict. art. Cæcum. [3] Principles of Surgery.
[4] Pathological Anatomy, 2d. edit. p. 632.
[5] *Edinburgh Medical and Surgical Journal,* vol. xxxi. p. 1, 1831.
[6] Medico-Chir. Transact. xx. p. 200, and xxii.
[7] *Northern Journal of Medicine,* July 1844.
[8] Diseases of Infancy and Childhood, 5th edit. p. 656. 1865.

plaint has in several recorded cases been present long before its nature has been discovered. Its earliest conditions are rendered manifest by the tumescence and dulness on percussion at the right iliac fossa. The circumscribed swelling may extend across to the umbilicus, and when such is the case Peritonitis is generally the accompaniment of other pathologic changes. The patient will complain of pain at the upper part of the thigh, and this has not the same freedom of motion as the other limb. It has repeatedly been found that there has been irregular action of the bowels, associated with colicky pains, which radiate from the iliac region. Dr. West says, that in children the bowels are mostly relaxed, and that pain in the stomach is an initiatory symptom ; and he also remarks, that the prominence in the right flank sometimes assumes that of an elongated tumour, which reaches from the ramus of the pubis nearly to the hypochondrium, and has a brawny hardness.[1]

When the ailment has for some time subsisted, lymph and purulent matter are deposited in the cellular tissues behind the cæcum, and so long as the strong iliac fascia prevents the escape of pus, a deep and irregular abscess is formed. The secretion at length most frequently passes through the cæcal parietes at the part uncovered by the peritoneum, as recorded by Copland, Duplay, and others. In some instances it is infiltrated into the cellular tissue in front of the iliacus internus, and effects an exit near the anus; or it may pass into the folds of the meso-colon, or make a sinus and be evacuated externally, as in examples related by MM. Husson, Dance, and Menière. Dupuytren knew it extend so high as the right kidney, and so low in the pelvis as to collect between the rectum and bladder. The perityphlic inflammation may be circumscribed and rather of the subacute than the acute type, with adhesion of adjacent surfaces. When the matter perforates the serous sac, diffuse and fatal Peritonitis ensues.

Peritonitis of Children.—Acute Peritonitis seldom occurs in infancy and childhood. It has been more frequently observed in young infants than in children several years older. Some have declared it may affect the fœtus ; in all such instances syphilis in the mother has been regarded as the cause, nor is it improbable that a general taint in the mother should impart disease to the child. Irritation of the digestive surface is more common in children than inflammation of the serous tunic. When Peritonitis does occur, it is generally as a complication or sequel. It may, however, be primary as well as secondary; it may be partial or general ; acute or sub-acute, and then pass into the chronic condition. When it appears it is mostly after one of the exanthematous fevers ; more especially after scarlatina or measles. Dr. West has not known more than half-a-dozen instances of acute general Peritonitis in childhood.[2] It has prevailed among young infants when exposed to deleterious external agencies. According to

[1] Diseases of Infancy and Childhood, 5th edit. p. 657.
[2] Ibid. p. 654.

M. Thore,[1] at the Hospice des Enfants Trouvés, at Paris, six per cent. of the infant mortality was from acute Peritonitis. It usually came as the complication or sequel of some other ailment, and no child above ten weeks was attacked by it. The fatal end was generally before twenty-four hours. Of sixty-three inspections in no case was there pus, but in all a greater or less amount of serum on which flocculi floated, and the intestinal coils and solid viscera were adherent. In seventeen out of the sixty-three, erysipelas had preceded the Peritonitis. Pleuritic effusion was discovered in a third of the examples.

The usual symptoms are pain in the bowels, which at first resembles common stomach-ache. It alternately subsides and returns, and there is mostly diarrhœa. In the course of a few days the pain becomes more fixed, and the child frequently complains of pain in the right side, and if old enough he indicates the locality by putting his hand on the cæcal or umbilical region. The pyrexial phenomena are proclaimed, the little patient looks haggard, he is restless and continually alters his position; pressure over the part makes him cry, and the abdominal muscles are tense. He lies on his back, often with legs extended, and the sickness is not so urgent as in the adult. According to Dr. West, when the affection is of cæcal origin, the right leg is often drawn up and the left extended.

Dr. George Gregory a long time ago described a form of marasmus, which he believed to be primarily disease of the peritoneum, and which he conceived to differ from what Pemberton terms "irritation of the intestines," and the kind of marasmus originating in the mucous membrane.[2] From being met with in scrofulous children, and an "imperfect kind of pus" being produced, he named it scrofulous inflammation of the peritoneum. He regarded it to be distinguished by abdominal tenderness, shooting pains which at the first come on in paroxysms, but at length increase in frequency and violence. The pain on touch is first localized, and then becomes diffused. Inspection revealed pus and agglutination of the viscera. But the account of this author applies more to chronic than acute Peritonitis. In acute Peritonitis of children pus is a rare consequence; when it is formed it gravitates into the lower parts of the abdomen, and is deposited in one or more collections or septa. It may be evacuated by pointing externally, as in empyema, or effect an exit by the bowels, and it is possible recovery may follow, but such is a possibility rather than a probability. When it occurs consecutively, as after some fever, and when the powers of vitality are lowered, turbid serum with a few floating flocculi is the common product, as I have already observed when speaking of the non-plastic type of the disease.

Complications.—This affection is often complicated with some other disease. It may be complicated with *gastritis*, a disease which

[1] De la Péritonite chez les Nouveaux-nées, in the Archives Gén. de Méd. August and September, 1846.
[2] Medico-Chirurg. Trans. vol. xi. p. 263.

rarely or never occurs in this country as an idiopathic affection, although it is said to do so in warm climates. The physician will, in nearly all cases, discover from the history of the case, or collateral circumstances, the cause of the inflammation. Gastric Peritonitis may be fatal without the contents of the stomach being poured into the serous sac, and without solution of continuity, especially when it occurs in a secondary form. But in such examples the inflammation is only limited. Sometimes tumours press upon the organ and inflame its serous covering, or the inflammatory condition may be there instituted by contiguity, as when neighbouring viscera, such as the liver, spleen, and intestines, are thus primarily diseased. Carcinoma, especially of the pyloric end, will sometimes, by the mechanical pressure, give rise to the result in question; when this happens the Peritonitis is generally of the more chronic description. In that form of ulceration of the stomach, which occurs mostly in young women, the general health is often not much affected. It is often in association with chlorosis, amenorrhœa, leucorrhœa, or sub-mammary pain, and the patient is apt to complain of a gnawing sensation at the epigastrium, accompanied with more or less of anorexia and vomiting. When the gastric peritoneum is rent or perforated by ulceration of the inner tunics, the pain is excessive, the powers of life are rapidly subdued, and death is inevitable.

When the peritoneum is inflamed in *hepatitis* it is generally in a partial manner, and it continues to be circumscribed unless extravasation of some description result, which is occasionally the case, and then the entire sac at once assumes the same morbid condition. Inflammation may begin in the parenchymatous structure and extend to the serous coat, and when such is the fact, the pain becomes more acute and defined, and the pyrexial symptoms are more pronounced. The right hypochondriac region is often full and tense, the normal lines of dulness are extended, there is pain on pressure and deep inspiration, and dyspnœa, coughing, and vomiting are frequent accompaniments. The patient cannot lie on his left side, and the recti muscles are rigid. When the convex surface is affected, the diaphragmatic investment assumes the same disease, and cough is a prominent symptom. The convexity may be inflamed without the appearance of jaundice. When the concavity is inflamed the stomach mostly becomes implicated, sickness is urgent, the gall-ducts are more or less obstructed, and jaundice, in greater or less degree, is a common result. When the parenchyma is alone inflamed, the pain is of a dull, aching character. When the serous tunic is involved the pain is sharp and acute. When lymph in considerable quantity is effused, the organ becomes adherent to adjacent surfaces, and if the albuminous exudation gravitate to the lower part of the abdomen, agglutination of the intestinal folds occurs. When hepatic abscess points to the surface, partial Peritonitis, by pressure, is induced. The effused lymph is protective from the worst consequence of extravasation. Hydatid tumours may, like abscess, excite adhesive inflammation. Cancerous growths occasionally pro-

duce subacute hepatic Peritonitis, but the symptoms are ill-defined and obscure. And the same remarks apply to tubercular masses in the capsule of the liver.

Sometimes we observe *acute splenitis* as an intercurrent complaint during the progress of intermittent fever. But, as I have more fully insisted in the article on Diseases of the Spleen, this organ is infinitely more prone to a chronic form of congestion. Sometimes, when during the cold stages the capsule becomes suddenly distended, such tenseness so stretches the fibrous and serous tunics as to usher in the inflammatory process; then pain of sharp and stabbing character, increased by pressure, is felt beneath the left costal cartilages radiating through to the back; the skin is hot, the pulse quick and hard, the urine high-coloured and scanty, the tongue furred, the bowels are confined, and if the under surface of the diaphragm has become affected, cough and dyspnœa are associated symptoms. The patient lies partly on his back with trunk curved to relax the abdominal muscles. Towards evening there is exacerbation of the symptoms. Post-mortem examination reveals the serous investment thick and reddened, and the organ united to neighbouring parts by albuminous exudation; and it is here not unworthy of remark, that in the peritoneal inflammation of this viscus, cartilaginous and ossific conversions are more frequent than in the peritoneal inflammation of the other solid abdominal organs.

In *enteritis*, when all the coats of the bowel are inflamed, the disease may commence in the mucous membrane, at first sickness and purging being urgent. In such cases colicky pains come on at intervals, and moderate pressure produces little or no uneasiness, and at this stage of the malady it is often difficult to form a correct diagnosis. If the complaint make progress, if the skin become hot and dry, the pulse quick, the face flushed, and pain be felt on pressure, it is of great practical importance to distinguish the kind of lesion to which the disease has advanced, because remedies which would relieve the colic would be absolutely injurious in inflammation. Instead of diarrhœa there is often constipation; thus it is when mechanical obstruction of the gut is the cause of its being inflamed, as in intussusception, and when tumours block up the passage, and vomiting of stercoraceous matters proclaims the inverted action of the bowel. The general and special signs of the peritoneum being inflamed are the same as those above described. In *children* the complaint is frequent during dentition, and it sometimes comes on as the sequel in eruptive fevers. Crude and indigestible articles of food in these little patients are often the cause. Its advent is marked by languor and peevishness, the child is restless and complaining, green mucoid stools emitting an offensive odour are voided, the cheeks become flushed, the belly tender, and all the conditions of peritoneal inflammation are superadded to a fever of the remittent type. And dissection sometimes exhibits the entire substance of a portion of the ileum presenting a gangrenous appearance in addition to the ordinary products of serous inflammation.

In *nephritis*—which is in the great majority of instances brought on by calculus in the pelvis of the kidney, blocking up of the ureter, some irritant drug, or some blow or external injury—severe pain over the loins following the course of the ureter on the same side, and, in the male retraction of the testicle, high-coloured urine and nausea and vomiting are common symptoms; and, as is occasionally the case when ischuria renalis supervenes, uræmic symptoms are apt to mask and obscure the otherwise more apparent features of peritoneal complication (*perinephritis*). The urinary bladder may be acutely inflamed (*cystitis*), the inflammation originating in the mucous membrane, and being extended to the muscular and serous coverings. It is caused by calculi, irritant drugs, retention, surgical operations, and external injuries, and the Peritonitis may be partial or general.

Hystitis is very rarely observed in the unimpregnated uterus; it may come on after menorrhagia by sudden suppression of the catamenia, long walks, wet and cold, and I have known it induced by the incautious use of topical applications. It is most frequent after delivery, and the fundus is the part mostly first affected. When the peritoneal investment becomes implicated the disease often assumes an alarming character. *Ovaritis* may be presented in one or both the ovaries without the uterus being inflamed; in the larger number of examples, however, it is the complication of general Peritonitis or antecedent uterine inflammation. Deep-seated pain in one or both of the pelvic cavities indicates the lesion, and when the peritoneum is affected the pain becomes exceedingly acute, and an aching, wearying sensation extends down into the groins and thighs. There is often frequent desire to micturate, and when the disease is continued to the posterior portion of the peritoneum the rectum is rendered irritable, and there is constant inclination to evacuate the bowels. Puffiness or swelling is sometimes seen over the ovarian region, and that part is most painful on the least pressure, and the sickness and vomiting are often distressing.

The comparatively recent establishment of that great surgical operation *ovariotomy*, more especially as practised in this country, has proved that the peritoneal sac can be laid open, and its inner surface exposed over a great extent, and for a considerable time, without the production of such fatal results as it was formerly believed would inevitably follow. It now appears, from a large accumulation of cases, that in a healthy subject, and especially in the unilocular tumour, and when there are no attachments, the peritoneum may be cut, and freely, without the consequent inflammation being always formidable.

There are some other affections with which Peritonitis is occasionally complicated. In pericarditis and pleuro-pneumonia it sometimes happens that the inflammation spreads to the peritoneum: but in such instances it is often extremely probable that a contaminated state of the circulatory fluids constitutes the predisposing cause, and that the irritation existent in one of the great cavities is readily transferred to another, and that an adjacent membrane of similar structure, and

under general predisponent circumstances, will take on the same morbid action. And, conversely, we know that Peritonitis often extends to the pleura, and it is not uncommon, as I have lately seen, to find hepatitis associated with dulness, moist crepitation, and all the other physical signs significant of inflammation in the lower third of the right thorax; and when the spleen is greatly enlarged, or in acute splenitis, the same conditions obtain at the base of the left lung; pressure and the proximity of like structures being the cause of such extension. In empyema the diaphragm may be rendered convex towards the abdomen, pushing down the abdominal organs, and friction and pressure induce Peritonitis; and in the enlargement of the liver or spleen, or an encysted kidney, or an ovarian tumour, this partition may be thrust up so abnormally into the chest as to press upon and excite the pleuro-pulmonary tissues to active inflammation.

MORBID ANATOMY.—The morbid appearances of Peritonitis are very various, being modified by a number of circumstances; such as the type, the primary or secondary character of the attack, the condition of the blood, the amount and kind of disease in the viscera, and more especially of the solid organs.

Before speaking of inflammatory change, it may be observed that serous membranes may be simply congested, presenting a condition analogous but not amounting to inflammation, and this hyperæmic state may be transient, temporary, or long-continued. When often returning or for some time existent it may give rise to excess of secretion, which is chiefly serous; nevertheless it may contain some coagulable matters, but their amount will be dependent upon the increase or diminution of the fibrinous and albuminous constituents in the blood. Such abnormal afflux of blood to this membrane may subside spontaneously, or there may be hæmorrhage into the sac, and such hæmorrhage may be passive or active,—it may be by transudation or rupture. Exhalation into the peritoneal cavity sometimes occurs, when a sanguinolent serum and an injected membrane are discovered. In visceral laceration considerable collections of blood of course may follow.

The gases generated in the cavity of the peritoneum are sometimes in great amount; they are in nearly all instances the result of cadaveric change and the decomposition of the secretions. In empyema, gases are produced when there is no solution of continuity in the pleura, and the same may result when there is pus in the abdomen and the peritoneum has maintained its integrity; but they may have their origin in ulceration of the intestines, or traumatic injury.

The first inflammatory change in the peritoneum is the loss of transparency and of that shining polished appearance proper to its healthy structure. This dulness or opacity is accompanied by diminution of the lubricating secretion, and Baillie, Bichat, and Knox affirm that the membrane becomes dry. But such dryness is more apparent than real, because when handled it feels moist and unctuous. The sub-serous vessels become injected, and may be seen through

the fine membrane in hair-like streaks, arborescent and ramified, or in a confused network, and when much crowded a velvety appearance is imparted. The degree or shade of redness depends upon the period of congestion, the kind of inflammation, and the condition of the blood. When the hyperæmia has for some time continued, or in sthenic inflammation, the hue is light red; when the congestion is but recent, or the inflammation of asthenic type, the colour is less vivid and may be darker and venoid.

With the progress of the disease, vessels in the membrane which were colourless enlarge so as to admit red-blood globules. At various points small sub-serous sanguineous effusions are seen in the shape of bloody puncta; sometimes these are so numerous as to exhibit a spotted or speckled appearance, or they may coalesce and form red configurated patches of various sizes. I have said that at the first there is diminution of the lubricating fluid. In the course of a short time (at periods differing according to certain conditions which obtain, such as the mildness or severity of the attack, the general powers of the system, and the like) this secretion is re-established, and if the malady end in resolution it manifests all the characteristics of the natural state; but if the complaint progress it is augmented in quantity and altered in quality. The free surface of the peritoneum is then bathed with a semi-transparent homogeneous fluid, and the sub-peritoneal tissue is surcharged with a sero-albuminous secretion, and frequently the peritoneum proper can be stripped off with undue facility. This infiltration, however, at length permeates the serous tunic, when it and the filamentous layer become so confounded that it is not easy to trace the line of union. Under such circumstances the membrane is not only rendered opaque, but it looks thick and tumefied, and if carefully examined it feels rough, has lost its lubricity, and close inspection detects a viscid albuminous deposit varying in thickness according to the duration and severity of the attack.

The new or morbid secretion which is effused soon separates into two distinct forms,—a thin and watery whey-like fluid, and a thick gelatinous, pulpy, or more solid portion; the former constituting serum, the latter coagulable lymph, or, as it is otherwise named, albuminous exudation or plasma. The relative proportions of the fluid and more solid parts vary in each individual instance. Sometimes we find no serum whatever, and sometimes the effusion consists almost entirely of serum, the only traces of the albuminous exudate being minute flocculi floating in the fluid and rendering it turbid. In the inflammation of metastasis and low types of Peritonitis the effusion is sometimes puriform, or absolutely purulent. In acute sthenic Peritonitis the lymphic deposit is great. It is thrown down on the free surface of the sac in various amounts according to the condition of the circulation and the violence of the inflammation. It may be a mere film or in a layer several lines in thickness. It differs in colour, being sometimes of greyish red, but is more frequently of a yellowish straw colour. When abundant, it lies in smooth or corrugated plates; it is

also found in honeycomb arrangement, in bands or bridles constituting bonds of union of varying thickness uniting the viscera, or it may be encircling the gut; it is generally seen in masses filling up the interspaces, and when lying between the intestinal folds it assumes an ill-defined prismatic configuration. The viscera are not only glued and matted together, but there is mostly more or less of adhesion to the parietal peritoneum. When a portion of the adventitious stratum is detached from the peritoneum, the coherent surface of the new product exhibits an irregular villous character, and it is speckled with small bloody puncta produced by torn capillaries, and the sub-serous tissue is ecchymosed. The new formation being at first villous, becomes smooth and more dense, and at length assumes a structure and qualities analogous to the true peritoneum.

If the exudation be submitted to the microscope new vessels are seen to permeate its substance, and more especially in the central portions. That they are connexions or prolongations of the peritoneal capillaries is beyond dispute, although we cannot always trace their continuous structure. It was believed by Hodgkin[1] that new vascular extensions are carried out into the exudation, and that subsequently towards the peritoneum they contract and become nearly or quite invisible. This author is of opinion that the delicate parietes of the extreme vessels give way, that minute quantities of blood are received into the exudation, and that such are the first beginnings of those minute cavities which are destined to become vascular.

It is quite evident that the plastic effusion is an irritant to the serous surface, because when deposited on one part of the peritoneum, and any other opposing part comes in contact with it, such readily takes on the inflamed condition; hence it becomes explicable, in one way at least, why Peritonitis is so liable to diffusion. According to the time which elapses after its production, and the vital powers of the organism, is the degree or completeness of the organization. From being a semi-fluid gelatinous substance it becomes more dense and solidified, the capillaries are more numerous, it contracts in bulk, its filamentous texture is more defined, and it enters into firmer and more intimate union with the organs or parts it covers or connects. Where there is much motion, it is sometimes disposed in a stringy or reticulated manner, and meshes are formed, filled with transparent fluid. Another morbid condition associated with these false membranes is that of serum or sero-purulent fluid being collected between the peritoneum and the false formation, until the latter is raised up and loosened from its attachments and set free in the sac. When these adventitious membranes remain firm and adherent, the original serous membrane beneath them disappears, and their surface assumes the characteristics of a veritable serous membrane, and it is difficult to distinguish the new from the old. The former secretes a lubricating serum, is influenced by the same kinds of irritation, is liable to become inflamed, and in its turn to throw out true inflammatory products.

[1] Lectures on Serous and Mucous Membranes.

The attachments effected by these formations may subsist through the remainder of life. They may be protective and conservative. In the suppurative stages, when abscess forms in the solid viscera, this adhesive inflammation is the method which nature observes for the harmless exit of pus. These bonds of union may continue with little or no inconvenience. By the lapse of time they become thin and contracted, and when health is re-established and the absorbents are active, they may partly or wholly disappear. Absorption begins with the subsidence of the inflammation, and, as Rokitansky[1] remarks, it must, as a matter of course, be influenced by the thickness, that is to say the permeability, of the deposit.

Before the time of the two Hunters it was not by pathologists generally allowed that serous membranes secreted pus without solution of continuity; in other words, without the presence of ulceration. Since then this fact has been universally acknowledged. It may be secreted from the inflamed peritoneum, or from the surface of those adventitious membranes which are formed in the cavity. William Hunter says it is generally thinner than that of an abscess, and the containing surface is more or less covered with a glutinous concretion or slough of the same colour as the fluid, in some parts adhering very loosely, in others so firmly that it can hardly be rubbed off, but still the surface covered with these sloughs is without ulceration or loss of substance.[2] Dupuytren and Villerme believe that the false membranes are concrete pus, and Rokitansky is of opinion that pus, under some inherent peculiarity, is a degeneration of plastic exudation. It is more frequently seen in the asthenic, subacute, and lower types of the complaint than in the sthenic. In the inflammation of metastasis, when the blood is contaminated, in parturient women, and in children, it is most common. The fluid may be puriform, purulent, or sanious. It may be yellowish green, or brown, or reddish. The peritoneum and sub-peritoneal tissue are much injected, and there is usually great infiltration of the tissues. In some instances it appears as if exuding from the entire inner surface of the peritoneum; in other cases it is associated with adhesions, and is discovered in distinct collections, bounded by organized septa, and resembling separate abscesses. It may be evacuated by ulcerative absorption through the abdominal parietes; by the same process it may pass into the digestive tube, the bladder, or vagina, or through the diaphragm into the thoracic cavity, or effect an entrance into the bronchi, or it may find a way of escape by the psoas muscle.

The pressure exerted by purulent collections is doubtless the main cause of ulceration commencing, but Craigie believes that in these cases sometimes ulceration may result without pressure, being merely the direct and obvious effect of inflammation. My colleague at the Tunbridge Wells Infirmary, Mr. Marsack, made (Sept. 18, 1865) an autopsy on the body of a young woman, on whom he had six week previously performed ovariotomy. The coils of the ileum were welde

[1] Pathological Anat., Syd. Soc.
[2] Medical Inquiries and Observations, vol. ii. p. 61.

together, and joined to the abdominal walls by organized adhesions. Between the layers of the great omentum were small, independent abscesses of creamy pus. In the lumbar region was a bounded abscess-like collection which contained half a pint of pus. At the sigmoid flexure ulcerative perforation was discovered.[1] Pressure, caused by a collection of purulent fluid, had been followed by ulcerative absorption of the tunics of the large bowel. When this secretion is effused in small quantity it may be absorbed, but if in large quantity and without opening, irritative fever is induced, the symptoms of pyæmia supervene, and it is then uniformly fatal. Sometimes adhesive inflammation in Peritonitis gives rise to very peculiar pathological conditions. The stomach and transverse colon have, in several instances, been glued together, and ulcerative absorption has effected a communication between them, so that the fæcal contents of the large bowel have passed into the gastric cavity, and thence been expelled by vomiting. Two or more coils of the ileum may be soldered together, and an intercommunicating passage established in the same manner. In such examples the disease has generally become chronic.

In the partial or localised forms of acute Peritonitis, when some foregoing visceral disease has extended through to the serous coat, and instituted inflammation in that tunic, we not infrequently see circumscribed depositions of lymph cementing neighbouring parts together while the remaining extent of the peritoneum is perfectly healthy. In hepatitis, when the convex surface is inflamed, strong adhesion is sometimes discovered. The spleen is in like manner united to the concave surface of the diaphragm, and the accretion may have assumed a cartilaginous or ossific character, the latter conversion being in that situation more frequently seen than in any other part of the abdomen. In simple ulceration of the stomach sometimes adhesive ulceration averts a fatal catastrophe by agglutination to one of the solid organs, or, as it has been repeatedly witnessed, by the production of an aperture into the colon, or sometimes into the duodenum; and, in a few rare instances, a canulous opening has been spontaneously made through the abdominal parietes, forming a gastric fistula. In malignant disease of this organ, most frequently seen at the pyloric end, there is much soldering together of the adjacent parts; the peritoneum is opaque and vascular, and the sub-serous tissue is greatly injected and infiltrated not only with carcinomatous deposit, but also with serous fluid. The duodenum, as before remarked, occasionally exhibits partial peritonitis from rupture, consequent upon ulceration of the mucous and muscular coats, as the result of extensive burns, but its serous investment is more frequently inflamed from the irritation and pressure resulting from cancer of the head of the pancreas. When the jejunum is found morbid it is almost always in connexion with the lesion of other organs. With regard to the ileum, what has above been said relative to the perforation of its peritoneal covering was descriptive of its morbid appear-

[1] Mr. Marsack's Hosp. Case Book.

ances. In phthisis sometimes protracted colliquative diarrhœa gives rise to ulceration in its mucous surface, but perforation in phthisis is exceedingly rare; it is, however, in this complaint occasionally beheld on or near the vermiform appendix. In chronic dysentery the colon may give way, and in such instances there is great destruction of the other tunics proper to the bowel. Such examples occur in those who have died after long residence in tropical climates, and in association with some form of hepatic disease—very generally with abscess of the liver.

In puerperal Peritonitis, according to Dr. Lee, the appearances of inflammation are sometimes confined to the uterus, but they are much more generally extended to other organs. The lymph is mostly thrown out in thicker masses upon the uterus than in any other situation, and this viscus seems to suffer in the greatest degree. In the sub-serous cellular tissue serum and pus are often deposited. The cellular tissue surrounding the vessels of the uterus where they enter and quit the organ, and that connecting the muscular fibres, is often surcharged with serum and purulent fluid.[1] The peritoneum becomes thick and vascular, more especially where it invests the uterus and pelvic viscera, and sometimes, when the malady is intense, the serum is mixed with blood, and pus is found in the pelvis. When death has rapidly followed, the lymphic exudate is semi-fluid, or the surfaces which have become agglutinated are readily torn asunder. The Fallopian tubes and ovaries are sometimes filled with pus or blood.

In the Peritonitis of children the abdominal viscera are found matted together and adherent to the abdominal walls. In some cases the viscera are covered with a thin greyish opaque covering, which feels soft and unctuous, and a turbid, reddish serum in which small flocculi are floating is effused in varying quantity. In that strumous affection which, according to Gregory, gives rise to Peritonitis, pus is secreted. And this physician asserts that sometimes the abdominal cavity will be abolished, the viscera being united in one mass, and everywhere adherent to the parietal peritoneum, the latter in all its duplications being thickened, and the soldered intestinal convolutions inter-communicating.[2] When the peritoneum becomes inflamed consecutively after scarlet fever, measles, rheumatism, or some other fever, an excess of serous effusion is discovered, the albuminous portion being inconsiderable or almost absent. The fluid is of whitish straw-colour or of dirtyish red.

DIAGNOSIS.—The more severe forms of acute Peritonitis are fully expressed, and the disease cannot well be mistaken; but in the sub-acute and more partial descriptions, when the disease is not a primary but secondary complaint, or a complication, it may be so masked, mixed up, and confounded with the symptoms of other morbid changes as to render the diagnosis very difficult. In all instances the physi-

[1] More Important Diseases of Women, p. 24.
[2] Medico-Chirurg. Transactions, vol. xi. p 266.

cian should pay marked attention to the history of the case, as well as to the objective and subjective symptoms, because there are affections which when superficially reviewed simulate this complaint, and it has not infrequently happened that the ignorant or off-hand practitioner has fallen into grave error. The diseases which it most resembles are gastritis, enteritis, colic, rheumatism, neuralgia, hysteria, obstruction of the gall-ducts, renal calculus, and lead-poisoning. With respect to *gastritis*, it is in this country, as I have before observed, rarely or never met with as a purely idiopathic affection. Abercrombie means by this term inflammation of the mucous membrane, and it is in such sense that it is now employed. When the mucous coat takes on this morbid state there may be pain on deep pressure, the sickness is urgent, the thirst distressing, and fluids are constantly ejected. It can almost always be traced to some exciting cause. In Peritonitis there is more difficulty in the etiological conclusion, and in the latter the pulse is smaller and more wiry. The inflammation may commence in the digestive surface and extend to the peritoneal investment, and it then, of course, becomes partial Peritonitis. It occasionally occurs when the gastric portion of the peritoneum is roughened by lymphic exudations that auscultation can detect some friction sound; but this, however, is seldom heard. In the great majority of cases gastritis is referrible to acrid and corrosive poisons. Haller knew it produced by the patient having taken cold water when he was heated. It is frequently very difficult, often absolutely impossible, to diagnose Peritonitis from *enteritis*. Inflammation may begin in the mucous membrane and implicate the peritoneum, or Peritonitis may at length involve all the coats of the bowel, when both diseases obtain. The vomiting is more urgent in enteritis, the bowels are often obstinately obstructed, and gangrene is sometimes the result. The pulse is of better volume than in Peritonitis, and as the rule the patient does not complain of so much pain. In Peritonitis, partly owing to the involution of the parietal peritoneum, the pain on pressure is more acute and superficial, the patient is more averse from motion, the respiration is more thoracic, and the features are more collapsed.

In *colic*, which may be from simple flatulence, the pain and distension may be severe, and even the face may be an index of suffering. When there is very great distension pressure may increase the pain, but more commonly pressure relieves rather than augments it; the circulation is little if at all affected, and there is no symptomatic fever. Frequently constipation and vomiting are associated with other symptoms; the patient complains of a twisting, wringing pain at the umbilicus, which comes on paroxysmally, and there are intervals when the suffering is inconsiderable or absent. This condition of colic is, when regarded alone and as simple colic, not an important affection, but it sometimes comes on as the herald of a more grave disease, and ends by the development of inflammatory symptoms. In *colica pictonum* there is no apparent obstruction of the bowels, although there are the common symptoms of ordinary colic. There

are constipation and abdominal pain, even violent pain—*dolor
atrox*—but there are other symptoms, such as pain in the head and
limbs, a blue, leaden line in the gums, and loss of power in the hands
and fore-arms, and the patient is either a painter, or investigation
discovers that he has in some way been subjected to lead poisoning.
The abdominal muscles in *rheumatism* sometimes are rendered so
excessively painful that moderate pressure causes great suffering, and
notwithstanding that examples are occasionally observed in which
acute Peritonitis has thus supervened, yet such instances are very
exceptional, and ordinary observation will generally prevent any
mistake in diagnosis.   Negative facts will be our chief guide.   In
such cases the circulation is little affected, the pulse is large and full
but not frequent, sickness and vomiting are not present, the counte-
nance has not the pinched, anxious expression which it assumes when
the peritoneum is inflamed, and if the abdomen be carefully examined,
the tenderness will be found most severe at the origins and insertions
of the muscles; lastly, it will be shown upon inquiry and examina-
tion that rheumatism has recently obtained, or that its symptoms are
still present in other parts of the body.

Neuralgia is another affection which mimics Peritonitis.   The
pain is described as a tight girdle or ligature passing round the
body, and imparting a feeling of constriction; it traverses the course of
the genito-crural nerve, percussion on the spinal processes detects
some tenderness, and the legs and genito-urinary organs are often more
or less affected; again, there is the absence of tympanites, pain on
pressure, quick pulse, facial collapse, and other phenomena so expres-
sive of Peritonitis, and which I have in detail described above.   In
that protean malady *hysteria*, which mocks this as it simulates so
many other affections, the patient is apt to complain of increased pain
almost before the hand has really touched the abdomen, and when it
does touch it, the pressure does not, as in Peritonitis, augment it.   The
pulse is natural, the tongue clean, and the countenance does not bear
the impress of severe and acute disease.   The breathing is not thoracic,
the legs can be extended, the decubitus is not dorsal, and borborygmi
and intestinal flatulence are often present; again, upon inquiry, it
will not infrequently be found that large quantities of pale or colour-
less urine have been voided, that the uterine functions are at fault, or
that some ill-defined spinal symptoms obtain.   A comparison of the
leading features common to the two affections will leave but little
doubt as to the true nature of the ailment.

In *obstruction of the gall-ducts* from calculi, inspissated gall,
tumours, spasm, and other causes, the pain is paroxysmal, often ex-
cruciating; and with the passage of the obstructing body, and the
restored patency of the canal, the suffering at once subsides.   There is
no pyrexia, the heart's action is little or not at all accelerated, nor is
there distension or abdominal tenderness.   In addition to such
negative there are positive facts; the symptoms of biliary disturb-
ance are mostly present, the alvine dejections are often light-coloured,

the urine is dark and porter-like, the conjunctivæ are yellow, the skin is tawny, and the pain is localized beneath the margin of the right false ribs. In *renal calculus* the pain radiates from the back round to the abdomen, it comes on suddenly, courses down the direction of the ureters, in the male produces retraction of the testicle of the same side, and shoots down the thigh, when for a shorter or longer interval it declines or entirely subsides, and bloody urine is a common accompaniment.

In puerperal Peritonitis the *after-pains* are associated with contracted, not relaxed uterus, which is the fact in Peritonitis; they gradually diminish, and in thirty or forty hours have become much less in force and frequency. Inflammation of the peritoneum commences at the ordinary date of the after-pains' decline. The remedial agents which relieve hysteralgia do not arrest acute Peritonitis. *Ephemeral fever* is distinguished by its brevity, its milder aspect, by the mammæ remaining of normal size, and those serious conditions which mark the advent of an inflamed peritoneum are wanting. Lastly, in speaking of the diagnosis of this affection, it must be borne in mind that under grave cerebral disease, when nervous sensibility is obtunded, the peritonitic symptoms may be rendered very obscure, and under such conditions diagnosis may be impossible.

PROGNOSIS.—The opinion to be arrived at relative to the result of this disease will be modified and determined by a variety of considerations, and in every case a different array of facts will be presented, all the bearings of which should be carefully scanned. The asthenic is less auspicious than the sthenic type, and when it is the inflammation of metastasis the chances of recovery are less. In *unfavourable* cases, in despite of the best-ordered means of treatment, there is a progressive aggravation of all the cardinal symptoms; the pain does not decline, nor do the distension and the tenderness abate; the breathing is more hurried, shallower, and entirely thoracic, the pulse becomes thready and intermittent, the sickness is excessive, the bowels are generally confined, distressing singultus supervenes, the surface becomes cool, is clammy and relaxed, the legs and feet are cold, the patient falls down in bed with knees drawn up, lies on his back, the Hippocratic countenance is more marked, and often the mind is clear to the end. He sinks by asthenia. In those instances when we can prognosticate a *favourable* termination, there is remission of pain and tenderness, decline of the distension, the sickness comes on at longer intervals, and at length abates; the pulse is slower and fuller, the temperature of the body equable and warm, the respiration is not so quick, and the diaphragm descends lower down, and the patient can turn on his side. When we have reason to believe that there is perforation of the bowel, rupture of the liver or spleen, the urinary or gall-bladder; when we suspect the evacuation of an abscess, or the effusion of blood, our prognosis must be unfavourable, and recovery under such conditions is well-nigh hopeless. In the consecutive form,

when the strength has been undermined by a previous malady, the probabilities of a fatal issue are great. In puerperal Peritonitis antecedent hæmorrhage and the amount of exhaustion induced by parturient efforts would influence our decision.

TREATMENT.—In every example of acute peritoneal inflammation, the remedies should be prescribed with a just reference to the emergencies of each particular case, because no trite and exact rules can be given admissible of universal application. The date of the disease, the powers of the patient, the kind of pathologic action going on, and the antecedent circumstances so far as they can be ascertained, in conjunction with other facts, must needs modify our resources, and be suggestive in the selection of those agents which are accounted as the most effective auxiliaries in combating the affection. That this disease, like many other ailments, when seen at the outset, and treated according to science and experience, can be guided and carried to a successful termination is of such every-day proof as not to require being insisted upon here. And on the other hand, if its progress be unrestrained by ignorance or timidity, it soon passes beyond the control of the most vigorous handling and the nicest skill. It is eminently one of those complaints which does not admit of vacillation and delay, promptitude and decision of purpose being of paramount importance.

In an acute attack of inflammation of the sthenic type, in the strong and hitherto healthy, and especially those who have lived in the pure air of the country, our best ally is *blood-letting;* but it is by far the most successful when performed at the commencement of the malady —as soon as possible after the pulse has become hard and quick, the pain urgent, and the disease established. It is then, by making a decided impression upon the circulating organs, that there is the greatest chance of the inflammatory action being cut short, and of those morbid processes being arrested which so quickly follow the development of the affection. Nor should we be deterred from the use of the lancet by the mere *smallness* of the pulse, because it may feel constricted, hard, sharp, wiry under the finger, for with the free emission of blood it will increase in volume and become soft and more natural to the touch. Many authorities, and some of high reputation, have spoken of the number of ounces which ought to be drawn at a first, second, or even third depletion, but there is no just rule as regards quantity. One patient will bear a much greater loss of blood than another, even when the two cases seem to bear a close resemblance. Our real and only reliable guide must be the effect produced by the abstraction. An influence must be made upon the heart's action, and the patient should, if possible, be bled in the erect position. Abercrombie recommends one or two small bleedings at short intervals after the first in order to keep up the good results of the primary depletion. There is no doubt if ten or a dozen hours are allowed to elapse after the first use of the lancet, and before a second visit, that in such long interval

the pulse may recover its strength, the initiatory symptoms in full force return, and a larger quantity of blood will require to be lost. In a disease so perilous the patient should at the outset be seen every two or three, or at least every three or four hours. It is within the first twenty-four hours that blood-letting is of the most avail. When effusion has set in and progressed to some extent, blood-letting is more likely to be harmful than useful. In the young and the robust, in those of ruddy complexion and high arterial action, and those who live in the purer air of the country, bleeding is much better borne, and it may need to be repeated. The dwellers in urban communities, especially amongst the badly nourished and ill clad, such as present themselves at the hospitals of the metropolitan cities and large towns, very rarely, if ever, require general blood-letting, and when it is had recourse to, a smaller quantity is followed by the desired effect.

After the lancet has been used it is excellent practice to follow it up by *local depletion.* Cupping is of course, from the pressure it would give, inapplicable; but twenty, thirty, or even forty leeches at one time may be applied to the abdomen, and often with the greatest benefit. Fomentations, by means of flannels immersed in hot water, and wrung out as dry as possible, the heat and moisture being kept up by their being covered with a large piece of oiled silk, is good treatment, and the flow of blood can thus for some time be promoted; or a large linseed-meal and bread poultice, or a bran poultice, produces a soothing effect. In the use of these applications, however, care should be taken to constantly renew them before they become cool, and when they are discontinued a dry hot flannel of three or four folds should be placed upon the abdomen. Another very valuable mode of treatment at this juncture is the employment of terebinthinate epithems. Two or three dessert-spoonfuls of the spirits of turpentine may be sprinkled over the wet flannel, or a large piece of spongio-piline the size of the abdomen may be wrung out of hot water, and the turpentine in like manner sprinkled over it; and these may be repeated two or three times if the patient can endure the applications. I can bear testimony to the very excellent effects of the external use of turpentine, which I have very frequently in this mode recommended, and I believe it to be a most valuable remedy.

The late Dr. Sutton of Greenwich was the advocate of cold applications in abdominal inflammation. He used cold enemata, and cold cloths made wet with evaporating lotions, and, as he asserted, with great benefit. Abercrombie also recommends this method of treatment. "In a considerable number of cases," says this physician, "I have used with evident advantage the application of cold by covering the abdomen with cloths wet with vinegar and water, or even iced water. Injections of iced water have been proposed, and I think it probable might be used with advantage."[1] M. Smoler of Prague has recommended cold compresses often renewed, and laid on the abdomen, their application being desisted from as soon as the patient sleeps; but

[1] Pathological and Practical Researches, 3d edit. p. 173.

he never allows the patient to change them with his own hands.[1] Not having any personal experience of cold appliances, I shall therefore not do more than mention a remedy to the success or otherwise of which I can bear no testimony. It would to myself at least seem of doubtful utility in many cases, and one involving great risk in others, and I prefer what I believe to be equally efficacious, and certainly safer, namely, warm fomentations.

After the abstraction of blood a large dose of *opium* should at once be administered, and two or three grains may be given in urgent cases. It then not infrequently happens that the patient has a tranquil sleep, after which he awakes with less pain, a moister skin, and with remission of the symptoms generally. In those instances in which sickness and vomiting from time to time come on, opium often acts most beneficially. If we wish to influence the system by mercurials, one grain of opium and three grains of calomel may be taken every four or six hours, and mercurial frictions on the thighs and in the axillæ can at the same time be adopted by means of the linimentum hydrargyri, which is perhaps the most convenient preparation for this purpose; or two grains of calomel and half a grain of opium may be given every second hour, and the inunction being also used until some slight effect be produced on the gums. Another mode of administering opium, especially when the stomach is irritable and ingesta are rejected, is by enemata.   Thirty or forty drops of laudanum can be injected in two or three ounces of starch gruel, and such repeated according to the exigencies of the case. If the bowels should be loose and the rectum inclined to expel its contents, a suppository composed of a couple of grains of solid opium with a sufficient quantity of Castile soap or cocoa-nut butter to form a conical mass, may be introduced *per anum*, and such from time to time as the physician may deem desirable. The indications denoting benefit having accrued from the above-named remedies will be mitigation of pain, softer and fuller pulse, easier and slower breathing, more relaxed skin, and diminution of the abdominal distension; the face, too, will look calmer and more natural, and the patient probably give expression to a more comfortable feeling.

Vesication is another of our aids in guiding the malady to a favourable issue. It may be done by means of the ordinary emplastrum lyttæ, or by the acetum cantharidis, or the liquor epispasticus, which are considered to act with more celerity. A large blister has sometimes appeared to be of service, but vesicants should not be applied at the outset of the attack. They are most advantageous when the initiatory symptoms are on the decline, when there is not such high arterial action, and when the surface has become cooler. I have seen them do harm when applied too early. The blistered part may afterwards be dressed with savin ointment, by which means a modified and beneficial amount of counter-irritation can be continued.

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[1] Betz's Memorabilien, and Gaz. Med. Lyon, Nov. 16, 1865.

When the stomach is so irritable that scarcely anything can be retained, *hydrocyanic acid* in an aqueous mixture, with a little glycerine or mucilage added, is one of the best of remedies. *Effervescing draughts* with the bicarbonate of potash and citric acid are sometimes given, but the evolution of carbonic acid gas by distending the organ makes it contract upon itself, and the contents are again pumped up. There is another objection to their use; as tympanites always in greater or less degree obtains, the distension of the stomach pushes up the diaphragm still higher, and renders the respiration more difficult; and, again, the neutral salt which is formed, by acting as an aperient, is liable to increase the peristaltic action of the bowels, a result which should be most sedulously avoided. When the tympany is very considerable a *fœtid injection* consisting of two drachms of the tincture of assafœtida in half a pint or a pint of decoction of pearl-barley may be administered; or an ounce of the oil of turpentine, first being made into an emulsion with the yolk of egg and then mixed with the same quantity of barley decoction as before mentioned, can be injected. The oil of turpentine taken in doses of ten or fifteen drops in some emulsion or bland drink, or five or eight grains of the compound galbanum pill, every six or eight hours, are good measures for adoption. When such do not produce the desired effect, O'Beirne's long elastic tube may be introduced high up into the bowel and there allowed to remain, by which means incarcerated gases find a ready way of escape and much comfort is experienced. It is when this condition of tympanites subsists, and gives great distress after the inflammation has ceased, that such measures are useful. When we do not feel certain that the inflammatory action has subsided, and when vesication has not removed the cuticle, terebinthinate embrocations are likely to be of service.

Constipation is another circumstance which in these cases generally obtains. A right and rational consideration of this matter is of cardinal importance, because the very wrong notion is sometimes entertained that the bowels must be moved, and under this erroneous reasoning drastic purgatives have been given, producing, as they were said to do, much mischief. The physician should bear in mind that the constipation is not the cause but often the *effect* of the inflammation, and that the indicated mode of procedure is first to subdue the inflammatory action, when in due time restoration of function will follow. To allay and mitigate peristaltic action—in other words, to give rest to the parts in a state of lesion—is to carry out the same principle observed in enjoining the disuse of a torn muscle, and in peremptorily excluding light in the treatment of an inflamed eye. If it is believed that there is great accumulation in the colon, an enema with olive oil and half an ounce of the spirits of turpentine in decoction of barley may be administered by means of the O'Beirne tube, and such may be repeated if deemed necessary; but there is benefit in frequently having recourse to this remedy in order to keep up gentle action of the intestines. To give purgatives by the mouth is often to set up or augment the irritation in the gastric

mucous membrane, and by increasing the peristaltic action in the bowels to aggravate the disease. The contents of the intestines are often but soft and pasty matters, and then their presence can do no harm. There is a far greater liability to error in being too solicitous respecting the movement of the bowels than in leaving them to the efforts of nature.

Diaphoretic and *diuretic* medicines are to be used with the foregoing. The acetate liquor of ammonia, the ætherial spirits of nitre with camphor julep, form a good mixture, and tend to keep the skin and kidneys in the performance of their functions. Small quantities of strong beef-tea or farinaceous food are to be given at intervals. Smoler of Prague gives a little broth once or twice daily, and as little drink as possible while the activity of the disease continues. Urgent thirst may be allayed by pieces of ice being put into the mouth.

Such, then, is the line of treatment to be pursued in the *sthenic* or more flagrant forms of inflammation of the peritoneum, but they are not often met with, and constitute exceptions rather than the rule. It would be out of place here to enter upon that troubled question, the change of type in disease, but certain it is, whether from agencies operating from without, or from causes originating in the organism itself, that depletion in this disease is very rarely warrantable in the way in which I have described; nevertheless it would be wrong to pass into that extreme of inertness which has of late become but too prevalent, for, as I believe, moderate blood-letting in rightly selected cases is yet, despite the conflictions of controversy and the caprice of fashion, a valuable remedy.

As observed, by far the greater number of cases of Peritonitis presented to our notice are of the *asthenic* type—in that adynamic state of the system that will not bear lowering, and in which the general strength should be husbanded, not destroyed: for instance, in such examples as are consecutive upon or the sequels of some foregoing malady, when following the eruptive fevers, when metastatic of erysipelas, when the complication of albuminuria, when it occurs in perforation of the bowel in enteric fever, in the bursting of a mesenteric gland, in phthisis abdominis, in those occult blood changes which affect general nutrition, as in cancer, struma, and the climacteric period, or cirrhosis and cardiac disease, and in contamination of the fluids, as in pyæmia and puerperal Peritonitis. When we have to treat it as related to such conditions, our remedial measures must be resolved upon with great modification. *Opium* in the asthenic form is the chief agent, and Drs. Graves and Stokes were amongst the first physicians who gave this drug very largely. An impression decided and speedy must be made upon the nervous and sanguiferous systems, and in such lies our main hope of arresting the disease. It should be given in large doses and the effect kept up in full and apparent manner, but not to the induction of narcotism. Two or three grains may at first be prescribed, and a grain every four, three, or even two hours afterwards. Some in very urgent cases give half a grain, or

even a grain, every hour. But in these perilous attacks of illness the patient should be frequently visited, and the physician should cautiously watch the effects of the remedy. Narcotism will be produced much sooner and with a far less dose in some persons than in others. If there be much sickness laudanum injections should at short intervals be administered, instead of giving the drug by the mouth. In cases of great prostration and debility, quinine and camphor may be conjoined with the opium. In *perforation*, when the contents of the bowel are liable to be extruded into the serous cavity, and when lymph is thrown out, by which means the conservative attempts of nature are to seal up the orifice and mend the breach, to subdue and still the action of the part is everything. Motion implies the pouring out of the intestinal matters, the removal of the lymphic plug—in other words, a fatal issue. To paralyse the bowel for a time is the aim, in order that reparation may be favoured. In these particular cases I would not give mercurials by the mouth. If they were to increase the flow of bile, and thus augment the peristaltic action, they would do incalculable harm. Inunction, as above recommended, might be used until the gums became slightly affected. It is far better to depend upon opium. In perforation there is sometimes very great tolerance of this drug. Murchison has known so large a quantity as sixty grains to be given in three days with impunity. In traumatic wounds, in the operation for hernia, and in paracentesis abdominis the same kind of treatment should be followed. Fomentations, turpentine stoups, or a large poultice may at the same time be employed. Subsequently vesication may be ordered—and such repeated according to circumstances.

In that kind of Peritonitis complicated with Bright's disease, the primary complaint should be more regarded than the intercurrent affection. Salivation is to be carefully avoided; diaphoretics, warm cataplasms, rubefacients to the loins, warm baths, the hot-air bath, vesicants, and nutrients are then indicated. When the acute symptoms have subsided, the compound jalap powder and Dover's powder may be given. When the attack follows the exanthemata, is metastatic of erysipelas, or connected with pyæmia, mercury is inadmissible.

In *puerperal peritonitis* the treatment is often difficult and doubtful, and it should earnestly be borne in mind that it is frequently associated with or consecutive upon an altered or vitiated condition of the blood. If the power of the pulse warrant the lancet, bleeding, to be of benefit, should be done *early*. If deferred it is likely to do harm. The best authorities are emphatic on this point. Dr. Ferguson asserts that to be beneficial it must be employed within the first twenty-four hours, and that in the second stage of the disease it often produces a rapidly fatal result. Churchill is of opinion that when the remedy is admissible the time for its beneficial use is very limited, and he has seen no good from its employment after the first twenty-four hours. The first-named physician in doubtful cases gave ten grains of Dover's powder, and covered the abdomen with a linseed-meal poultice, which from its thickness would keep warm for four hours. At the expiration

of that time, if the symptoms were not relieved, ten grains more of Dover's powder and another poultice were prescribed. If in other four hours from this second medication the malady did not yield, he had recourse to depletion. Sometimes when the pain is great and the pulse tolerably firm, two or three dozen leeches at once applied and followed by fomentations give good results. In the majority of cases, measures will be required which have previously been described as suitable to the asthenic type of this inflammation.

In the *Peritonitis of children* those general principles are to be aimed at which have already been given. It need scarcely, however, be more than mentioned here that these little patients always require their maladies to be managed with a gentle hand, and most especially in the use of depletion and opiates. These remedies with them are very uncertain in their effects, and sometimes produce a far greater impress upon the general powers than calculated upon by the practitioner. The age, the history of the case, and the cardinal signs will be our guide, and our measures should be modified according to the facts and exigencies of each particular instance. In the sthenic types, leeches, calomel, and if the age permit, carefully regulated doses of opium, linseed-meal poultices, terebinthinate epithems, warm baths, and injections are to be used. When the affection comes on as the sequel of one or other of the eruptive fevers, if we believe it to be traceable to some constitutional malady, some depravity of the organism, depletion and antiphlogistic means will be unwarrantable; then mercurial alteratives, small opiates, fomentations, warm baths, and counter-irritation will be the best measures. When the little patient tides over the more perilous days of active disease, and the case drifts onwards towards the more chronic condition, and when we find that there is effusion, counter-irritation, and mild mercurial alteratives should be given, and during convalescence the iodide of potassium with decoction of sarsaparilla, the syrup of the iodide of iron, or quinine with the tincture of the perchloride of iron, often produce excellent effects. In the strumous diathesis cod-liver oil may be prescribed.

It has in this article been previously pointed out to the reader that Peritonitis not seldom occurs in a partial manner, and as a *complication* arising in the course of some foregoing disease, as when an antecedent malady, first instituted in some organ or organs covered by the peritoneum, is at length extended to it. For instance in hepatitis, when the convex surface is the seat of lesion it remains circumscribed; or the inflammation may be extended through to the pleura, and pleuro-pneumonia result, as in a case which I recently witnessed. It is then quite clear that our remedies should be addressed to the viscera involved, as well as to the serous membrane. In acute splenitis the turgor of that viscus should be relieved, or it would be vain to try to mitigate the peritoneal symptoms, which have their origin in the stretched, tense, irritated condition of the capsular coverings. In the liver affection we should as soon as possible bring to bear the influence

of mercurials; but in diseases of the spleen mercurials are most improper, and would do harm.[1] It is incontestable then that our diagnosis must be rightly formed, or our practice will be incorrect. In diarrhœa and dysentery, when associated with an inflamed peritoneum, it is needful at once to control the excessive action of the bowels, and when such is subdued, the irritation extended to the serous membrane is likely to be subdued also. Opiate enemata, fomentations, the compound ipecacuan powder, and counter-irritants are the best measures. It has been remarked that the right iliac fossa is often the seat of pain, the disease being located near the cæcum, and it sometimes happens that the impaction of indurated fæces has much to do with setting up the inflammation. Large bland enemata, by unloading the great bowel, are in such cases of excellent service. When the sexual and urinary organs are first affected and Peritonitis becomes superadded, the primary disease should be held in view, and by its mitigation or removal the consecutive complaint will be benefited. From all, then, which has been said, it is obvious that in the treatment of every case the successful issue will greatly depend upon a clear and correct conception of the nature of the ailment, and a right interpretation of those symptoms which indicate the particular kind of morbid changes which obtain.

When the more acute stage has passed over, and those remedies suited to the earlier period of the attack have been employed, small doses of opium may still be given in combination with quinine or some of the bitter infusions. The various preparations of iron are of great value, and perhaps the tincture of the perchloride is the best. It is safest to defer as long as possible the use of aperients, and in preference the gentle action of the bowels should from time to time be promoted by bland enemata. When the active state of the affection has quite ended, an occasional dose of grey powder with rhubarb and the bicarbonate of soda may be given. Terebinthinate and other stimulant embrocations can be applied to the abdomen when there is effusion, and a flannel bandage round the body, so as to ensure moderate and well-regulated pressure, is another mode of favouring absorption.

The *diet and regimen* during convalescence are of great importance. At the first soups and farinaceous food are to be allowed, and for some time solids should be interdicted. Arrow-root, tapioca, the Indian cornflour, with milk, are nourishing; and veal or chicken broth with the crumb of bread may be given; and in the course of time beef-tea with toast, boiled chicken, and pounded meat may be taken. When stimulants are needed, sherry, weak brandy and water, claret, and bitte ale may be allowed. Flatulent vegetables and acescent fruits should for some time be discarded. An occasional warm bath to keep the skin in proper action is desirable. When the patient shall have so far recovered as to be able to travel, change of air will generally expedite his restoration to health.

[1] See Art. on Diseases of the Spleen in this work, where I have expatiated on the ill effects of mercury on diseases of this organ.

TUBERCLE OF THE PERITONEUM.

By John Syer Bristowe, M.D., F.R.C.P.

Pathology.—The deposition of tubercular matter in connexion with the peritoneal membrane is of very common occurrence. For generally in cases of tubercular ulceration of the bowels, and certainly in all those cases in which the ulceration is extensive, gray granulations may be found in greater or less abundance studding those areas of serous surface which correspond to the areas of mucous ulceration. But tubercular formations of this kind seldom show any tendency to spread, and are rarely productive of appreciable mischief. They are for the most part, indeed, purely local phenomena.

There are other cases, however, far less common yet still not infrequent, in which the tendency to the deposition of tubercle is general throughout the serous membrane, and in which ulceration of the bowel is evidently not the starting-point of the peritoneal affection, and indeed is often altogether absent. To these, which were formerly known as mere varieties of chronic peritonitis, the name of Tubercular Peritonitis is now very often given. They are characterised not only by the comparative severity and extent of the peritoneal affection, but also by the fact that the symptoms of this affection are usually well-pronounced, and sometimes indeed are paramount.

Tubercular Peritonitis, like tuberculosis generally, may occur at any age, but is probably most common in early life. Out of 48 cases extracted from the records, for a limited period, of St. Thomas's Hospital, 3 were under ten, 14 between ten and twenty, 13 between twenty and thirty, 9 between thirty and forty, 7 between forty and fifty, and 2 between fifty and sixty. But in correction of these figures it must be recollected that children under ten are admitted in small proportion into general hospitals. Out of the same number of cases, 26 were males, 22 females; but 222 tubercular males were admitted to 127 tubercular females, and proportionately to this number tubercular peritonitis was more frequent in the female than in the male, in the ratio of very nearly three to two. In two cases only was the tubercular deposit limited exclusively to the peritoneum. In all the others —namely, in 46 cases—there were tubercular deposits in other organs and generally in several other organs. In 42 there was tubercle the lungs; in 25, in the intestines; in 25, in the pleuræ; in 20, in the spleen; in 14, in the bronchial glands; in 11, in the kidneys; in 1

in the mesenteric glands; in 9, in the liver; in 8, in the brain; in 4, in the uterus and Fallopian tubes; and in 1, in the pericardium. But taking into consideration the relative frequency with which the several organs just enumerated are the seats of tubercle, a very different numerical relation than that just given becomes apparent between tuberculosis in them severally and tuberculosis of the peritoneum. Thus tubercular disease of the peritoneum was present in (to disregard fractions) 74 per cent. of cases of tubercle of the pleura, in 53 per cent. of cases of tubercle of the spleen, in 46 per cent. of cases of tubercle of the kidneys, in 44 per cent. of cases of tubercle of the brain and of the uterus and Fallopian tubes respectively, in 39 per cent. of cases of tubercle of the liver, in 37 per cent. of cases of tubercle of the bronchial glands, in 33 per cent. of cases of tubercle of the pericardium, in 29 per cent. of cases of tubercle of the mesenteric glands, and in 12 per cent. only of cases of tubercle of the lungs and of tubercle of the intestines severally. It may be worth while to add, that out of the 46 cases in which there was tubercular deposit in other organs besides the peritoneum, the most serious lesion was in 12 the tubercular disease of the peritoneum; in 15, that in the lungs; in 8, that in the brain; in 3, that in the pleura; and in 1, that in the intestines.

Peritoneal tubercles present much the same characters as tubercles occurring in other parts. They are sometimes miliary, or in the form of minute roundish spots, varying from mere points up to the size of a poppy-seed, and having an opaque white, or greyish or yellowish aspect. Sometimes they form rounded or lobulated masses, from the size of a tare up to that of a hazel-nut, presenting for the most part an opaque buff-colour, studded often with black points or patches, and exhibiting a cheesy aspect and consistence which are modified by the more or less abundance of fibroid material which invests and permeates them. Sometimes again, but much more rarely, there are found, lying between organs which are adherent, tubercular laminæ of considerable thickness and extent. Peritoneal tubercles exist rarely, if ever, independently of the effusion of lymph, and indeed rarely, if ever, are formed otherwise than in the substance of such adhesions, although they may subsequently in the progress of enlargement involve not only the peritoneum itself, but the tissues which are subjacent to the peritoneum. There is probably no essential distinction between the miliary form of tuberculosis and that in which the tubercles form masses of larger size: the former, however, are most frequently found in cases of acute progress, the latter in cases which are chronic; the former, moreover, are generally discovered thickly-set and innumerable, the latter in comparatively small numbers. In cases of miliary tuberculosis, indeed, the peritoneal surface is mostly found covered with a layer, of various thickness, of greyish, transparent, adherent and toughish lymph, which not only invests the abdominal organs, but renders them more or less adherent to one another. And in the substance of this lymph the tubercles are disseminated as opaque

grains, which may be separated with the lymph from the subjacent peritoneal surface. This condition may be general, or it may be limited to certain regions, and not infrequently when thus limited the parts affected are studded with filaments of lymph, in which miliary tubercles may be recognised. In the other form of the disease, the peritoneal surface is covered with lymph which has assumed the form of connective tissue, and the adhesions between organs are formed of tough fibrous bands. And it is in this tissue, and especially among these bands (sometimes forming the centre of a kind of knot, sometimes forming flattened masses between closely united surfaces), that the large masses of tubercle are for the most part found. It is this form of tubercle which occasionally invades the intestinal walls, and leads to perforation of the bowel from without. In association with the deposition of peritoneal tubercle, the various accompaniments and sequelæ of common inflammation manifest themselves generally in a greater or less degree. Thus, there is often patchy and streaky redness, often fibrinous effusion which is not visibly tubercular, and often effusion of serum; sometimes there is suppuration, and sometimes again hæmorrhage into the peritoneum. The most important of these, from its frequency, is undoubtedly the effusion of serum. Indeed tubercular disease of the peritoneum is a common cause of ascites. It is probable that most cases in which tubercle exists on the peritoneal surface prove fatal sooner or later, either from the direct effects of the peritoneal disease or from the effects of tuberculosis in other organs. Yet there can be no reasonable doubt that recovery sometimes takes place. For not only does our knowledge of the progress of tubercle in the lungs justify us in this inference, but we not infrequently meet with cases of recovery from symptoms which we have the strongest reasons to regard as dependent on tubercular peritonitis, and still more, we occasionally detect in the abdomens of persons dead of other diseases signs of old peritonitis, together with the presence of earthy nodules such as result from the drying up of tubercle.

SYMPTOMS.—The symptoms which attend the progress of peritoneal tuberculosis present much variety, and are often vague and indefinite. Often, indeed, and not only in those cases in which the peritoneal affection is slight, or in those in which it is as it were overshadowed by the preponderance of disease in other parts, but in those cases even in which it is the predominant or sole affection, they fail to indicate clearly the peritoneum as the seat of any disease. Further, they are so generally complicated with the symptoms which are due to co-existing tubercular disease in other organs, especially in the lungs, pleuræ, and intestines, that it is impossible altogether to dissociate them from these latter.

Most cases, however, of tubercular peritonitis, in which there is obvious indications of abdominal disease, may be arranged, somewhat roughly perhaps, in two classes: the first, the acute class, in which

the symptoms bear a considerable resemblance to those of enteric or of so-called "remittent" fever; the second, the chronic class, in which the symptoms correspond for the most part with those of "chronic peritonitis." In the acute form of the disease, the patient, sometimes in the midst of perfect health, more often however after some indefinite period of languor and loss of flesh and strength, begins to manifest febrile symptoms attended with remissions and indicated by heat and dryness of surface with quickened pulse, pains in the limbs and loins and head, diminution of the secretions, and perhaps drowsiness. At the same time the abdomen probably becomes somewhat hard and tumid and tender, and the patient complains of more or less pain in it. Generally also there is some disturbance of the digestive functions, dryness or furring of the tongue, thirst, loss of appetite, and nausea or sickness, with probably constipation or diarrhœa, or an alternation of these conditions. And with no material change in these symptoms, perhaps, beyond that which is due to gradually increasing debility and emaciation and the gradual supervention of what are ordinarily known as "typhoid symptoms," the patient gradually sinks, and at the end of a few weeks dies. The distinctions between acute abdominal tuberculosis and enteric fever consist, as regards the former disease, partly in the absence of rash, the less constant disturbance of the bowels, the non-limitation of tenderness to the cæcal region, and the less definite duration of the disease, and partly in the occasional presence of characteristic complications, among which may be enumerated tubercle in the brain, pulmonary phthisis, renal disease with albuminuria, and the accumulation of ascitic fluid. It may be remarked, however, that even in spite of care the cerebral symptoms arising from tubercle in the brain may be mistaken in some cases for the delirium of enteric fever, and the symptoms of pulmonary tuberculosis may pass for those of the pulmonary affections which so commonly ensue in that fever; and further, that the liability in both cases to intestinal perforation and acute peritonitic symptoms furnishes an element of serious difficulty in reference to diagnosis. In the chronic variety of peritoneal tuberculosis, the disease sometimes commences with more or less typical symptoms of acute peritonitis, sometimes creeps on with the utmost insidiousness; but in both cases (in the one after the disease has become fully established, in the other after the acute initial symptoms have subsided) the symptoms gradually become more or less identical with those which have been described elsewhere as indicative of chronic peritonitis: symptoms which, with many variations and remissions and exacerbations, may continue for a month or longer, and upon which in most cases sooner or later ascites supervenes. It must not be forgotten that in the chronic, as well as in the acute affection, deposition of tubercles in other organs is apt to take place, and that in its course the presence of tubercles in the brain, lungs, bowels, or elsewhere may produce symptoms which may lead us or mislead us in our diagnosis; and that in this case, even more than in the other, there is liability to tubercular perforation of the bowel, and to larda-

ceous or other degenerative diseases of important organs, especially of the liver and the kidneys.

As examples of some of the many anomalous cases which do not by their symptoms fall very obviously under either of the above categories, I may here briefly quote two cases. A girl about twenty had been ailing for some twelve or fifteen months. She had been getting weak and thin, and had been suffering from attacks of severe sickness, coming on with some regularity every three or four days. The sickness was remarkable from the facts that during the three or four hours for which it lasted she would bring up as much as a couple of wash-hand-basinfuls of nearly clear fluid, that it was apparently independent of the ingestion of food, and that between whiles she had no symptoms of indigestion and had a good appetite. There was, further, no affection of the bowels, and no distinct abdominal enlargement or tenderness. These symptoms continued while she was under my care; but shortly after she came under my care, and then for the first time, a cough came on, consolidation was discovered under the left clavicle, and from that time pulmonary consumption made rapid progress. Her death, which was mainly caused by the pulmonary disease, occurred about three months after I first saw her; and at the post-mortem examination there was found, in addition to extensive tubercular disease of the lungs, very extensive peritoneal tuberculosis. The stomach and bowels were healthy. A young gentleman of two or three and twenty, who was at the time resident at Port Natal, became without any apparent cause subject to attacks of intense colic, in which he was compelled by the severity of the pain to throw himself down and writhe. He came over to England in consequence of the persistence of this affection. The attacks of pain still continued, coming on sometimes two or three times a day; but there was also some irregularity of the bowels. His illness lasted for about a couple of years, and he died then from emaciation and exhaustion. There was more or less general tuberculosis discovered after death; but the chief deposit was in connexion with the peritoneum. Occasionally the chief symptoms due to the presence of peritoneal tubercle are great obstinacy of the bowels, with gradually increasing emaciation and debility; and occasionally there is complete and insuperable obstruction. In some cases, ascites is the earliest prominent symptom, and it may continue the most prominent symptom, and then prove (as ascites from other causes often proves) the chief agent in causing death.

CARCINOMA OF THE PERITONEUM.

By John Syer Bristowe, M.D., F.R.C.P.

PATHOLOGY.—Carcinoma of the Peritoneum, using the term in its widest sense, is not infrequently met with. Taking for comparison the same period which furnished, from the medical wards of St. Thomas's Hospital, 349 cases of tuberculosis of which 49 presented peritoneal complications, there were 99 cases of cancer, in 22 of which the peritoneal membrane was affected. From these figures it would appear that while cancer of the peritoneum is less than half as common as tuberculosis of that membrane, it is considerably more common in reference to all cases of cancer than tubercle of the peritoneum is to all cases of tuberculosis.

There is probably no great difference in the liability of the two sexes to this disease; but there is no doubt, I think, that it is relatively less frequent in early life than tuberculosis. Of the 22 cases alluded to above, none occurred under twenty, 3 occurred between twenty and thirty, 4 between thirty and forty, 5 between forty and fifty, 5 between fifty and sixty, and 5 between sixty and seventy. In two cases the disease was apparently limited to the peritoneum, or had at most invaded the surface of organs invested with the peritoneum. It was associated in 11 cases with cancer of the stomach, in 10 with cancer of the liver, in 9 with cancer of the pleurae, in 7 with cancer of the lungs, in 6 with cancer of the mesenteric glands, and in 3 severally with cancer of the bowels, kidneys, and ovaries. More than half the cases, however, of cancer of the bowels were combined with peritoneal cancer; rather less than half the cases of cancer of the pleurae and stomach respectively were associated with it; and about a fourth of all cases of cancer of the liver, mesenteric glands, kidneys, ovaries, and lungs respectively presented the same complication. In 7 cases the peritoneal cancer was the predominant disease, in 10 cancer of the stomach, in one cancer of the liver, and in one cancer of the mesenteric.glands. It may be added here, that in speaking of peritoneal cancer, those cases have been excluded in which that portion of peritoneum covering a cancerous organ has alone presented indications of cancerous growth.

Carcinoma of the Peritoneum presents most of the varieties which carcinoma presents in other parts of the body; namely, scirrhus, encephaloid (with its sub-variety melanotic cancer), and colloid.

Scirrhus always commences in the form of flat, round, lenticular, hard, white spots, measuring perhaps on the average a line in diameter, which occupy the substance of the serous membrane, and though distinctly projecting from the surface, yet rather tend to invade and involve the sub-serous tissue. These are in the first instance scattered thinly or irregularly, but soon become aggregated in parts or generally, and then coalesce so as to form patches of various extent. The patches thus formed may be perfectly smooth on the surface, or may still present there traces of the mode in which they were originally formed; they rarely, however, form outgrowths, and pretty rarely invade subjacent organs; rarely, too, over the general peritoneal surface do they become more than a line or two thick, except when they involve duplicatures or processes of peritoneum. The latter involvement is indeed somewhat characteristic of the disease. The appendices epiplöicæ become converted into small hard masses, in which the cancerous deposit and the fat and other normal tissues become intermixed; the mesenteric and other like duplicatures become often similarly affected; and the great omentum, from the same cause, becomes contracted into a thick band, stretching transversely across the abdomen in the course of the transverse colon. Scirrhous cancer, in fact, as has long been recognised, tends rather to cause contraction of parts than outgrowths: and for this same reason has a special tendency not only to cause the contractions of loose tissues already adverted to, but to lead to obstruction of tubular organs, especially of the stomach, intestines, and larger bile-ducts. Encephaloid also in its early stage affects the substance of the peritoneum, and forms discrete nodular outgrowths, which are small and rounded, and differ from those of scirrhus not only in their greater softness, but also in their greater prominence. These are often indeed hemispherical, or even spherical or pyriform and pedunculated. In its further progress encephaloid presents great varieties. In some cases it seems, like scirrhus, to invade more particularly the substance of the peritoneal folds, and to involve also subjacent organs; and under such circumstances we find sometimes the mesentery converted into a thick, plicated, cancerous mass, with the cancerous growth extending from the mesenteric attachment over the surface of the intestines; or we find the greater or lesser omentum or the sub-peritoneal tissue of other regions affected in like manner, and forming a more or less distinct tumour. In other cases it tends rather to form outgrowths which are sometimes small and clustered, sometimes more or less distinct from one another and rounded and massive. In the former instance the whole peritoneal surface may be found beset with small lobulated or bunch-of-currant-like excrescences, and the great omentum may be converted into a huge loose mass of such bodies. In the latter instance the tumours, though more or less abundant, are isolated, and while many probably are small, others form rounded solid masses which may attain the size of a child's head. So far as I know, melanotic cancer always manifests itself in this latter condition. Colloid disease in its early stage appears for the most part in the form of groups of vesicles

which vary in fineness and have a close *primâ facie* resemblance to patches of eczema or herpes, or (if the fibroid element be abundant) in the form of slightly granular or delicately-reticulated patches. Later on, the vesicle-like bodies are often as large as a millet-seed or tare. The patches often become more or less elevated above the level of the surrounding surface, and spread sometimes in tortuous and anastomosing lines as though taking the course of the lymphatic vessels, sometimes by forming scattered, isolated, somewhat pedunculated growths. This disease, like scirrhus and encephaloid, tends in various degrees both to involve subjacent organs and to diffuse itself over the peritoneal surface. It always involves the sub-peritoneal tissue, which may attain in consequence very considerable thickness; and it extends thence most frequently to the muscular and mucous coats of the stomach and intestines, less frequently to the substance of the mesenteric glands, pancreas, liver, spleen, or other viscera. In the most extreme cases of the disease, nearly the whole of the peritoneum is affected; this membrane is then irregularly thickened, with lumpy excrescences here and there; the various duplicatures become especially hypertrophied; and the great omentum is sometimes converted into a huge lobulated mass, or is contracted, as it generally is in scirrhus, into a thick irregular transverse band. In all these cases the adventitious growth retains its original more or less distinctly vesicular if not gelatinous character; and generally, sooner or later, from erosion of its surface, the glairy fluid contained in its substance is discharged in some abundance into the cavity of the abdomen.

Other varieties of cancer, such for example as osteoid cancer, are probably always secondary, and are of such extremely rare occurrence as to be of no practical importance.

All forms of abdominal cancer are liable in a greater or less degree to various complications. Among which may be enumerated: peritoneal inflammation, with the effusion of lymph or pus, or the escape of blood; ascites; obstructions of stomach or bowels; involvement of the viscera, such as the liver or kidneys, or their excretory ducts; and perforations of the stomach and intestines or other hollow organs.

SYMPTOMS.—The symptoms of peritoneal cancer are necessarily very various, and often quite as easy to be misunderstood as those of peritoneal tubercle. Febrile symptoms, varying in intensity and liable to remissions, gradually increasing debility and emaciation, more or less uneasiness or tenderness or pain in the abdomen, with hardness and enlargement of the same part, disturbance of the functions of the alimentary canal indicated by dry and glazed or coated tongue, thirst, loss of appetite, with perhaps nausea and sickness, and by constipation or diarrhœa or alternations of both, are symptoms which are common alike to cancer and to tubercle and to mere chronic inflammation of the peritoneum. It is important, however, to bear in mind that obstinate constipation is a very

frequent accompaniment of this disease, and that much more frequently than in either tuberculosis or inflammation, death results from complete obstruction; also, that in a very large proportion of cases the stomach is involved in a greater or less degree, and that consequently the usual symptoms of stomach-cancer are very liable to be associated with those of the peritoneal affection; further, that in nearly half the cases there is cancer of the liver, not infrequently involving that organ through the gastro-hepatic omentum and Glisson's capsule, and that therefore obstruction of the bile-ducts and jaundice are of common occurrence; and lastly, that in the female there is frequent co-existence of ovarian and peritoneal cancer. The most important points, however, to which we must look for the formation of a correct diagnosis are, first, the presence of a growing tumour or tumours in the abdomen, and, second, the presence of similar disease in other parts. It need scarcely be said that cancerous tumours present all varieties of character; that they may occur in any region of the abdomen; that they may be moveable or fixed; that they may vary widely in size and shape; that they may be hard and resisting, or soft and almost yielding a sense of fluctuation; and that, especially when they are developed in the neighbourhood of the cœliac axis and superior mesenteric artery, they may pulsate as violently as many aneurisms do; and that hence, notwithstanding the important aid which their presence furnishes, they may be, and are not infrequently, confounded, at some stage at least of their progress, with circumscribed abscesses, or hydatid tumours, or floating kidneys, or even aneurisms. But in some cases where, although the cancerous disease is very extensive, the individual tumours are small, the presence of the peritoneal outgrowths may fail of detection, even when very careful examination has been made; and necessarily this difficulty of detection is always greatly increased when ascitic fluid is present. It is worth while to draw attention to the fact, that not infrequently when no other signs of tumour are distinguishable, the presence of the thickened and contracted great omentum, which has been shown to be common in scirrhus and in colloid disease, may be recognised as a more or less irregular transverse bar extending horizontally from under the margins of the left ribs across the upper part of the umbilical region to the neighbourhood of the umbilicus, and that this furnishes a valuable diagnostic sign.

It is impossible to lay down any rules with regard to the detection of concurrent cancerous disease in other organs; but it is obvious that in all cases in which there is any ground to suspect that a patient may be suffering from internal cancer, a careful investigation of all superficial and other easily accessible parts should be made; for not infrequently there may be found associated with the internal cancer, coming on before it, or appearing at a later period, cancerous nodules in the subcutaneous cellular tissue, cancerous growths of periosteum, or bone, or cancer affecting the uterus, mamma, or testis. Nor must it be forgotten that cancer of the pleuræ, lungs, and mediastinum,

cancer of the brain, and cancer of the kidneys, are all with different degrees of frequency apt to be associated with cancer of the peritoneum.

Treatment of Abdominal Tubercle and Carcinoma.—There are stages in many varieties of the diseases coming under the above heads, when, as has been shown, they may be readily mistaken for other affections of a less grave character than themselves; and when therefore it may be judicious to adopt the treatment, whatever it may be, which may seem most suitable for the more curable malady. But, assuming the fact of the presence of tubercle or of cancer to be known, the principles of treatment become exceedingly simple: they are, to relieve pain and discomfort by ministering to those symptoms which most distress the patient, and to support his strength by the judicious exhibition of food and stimulus, and by the use of medicines having a similar tendency. Abdominal pains may need to be relieved by the application of counter-irritants, or fomentations, or even leeches. Sleepless weariness and pain may require to be overcome by the use of opiates or other forms of sedative or narcotic medicines; and indeed, in the progress of cancer especially, these remedies are often the only ones that can be employed, and may have to be given constantly and in large doses. Nausea, sickness, diarrhœa, obstruction of the bowels will each in various cases call for treatment, but nothing special need be said in reference to them. That tonics, food, and stimulants, of such kind and in such quantities and at such intervals as the condition of the patient renders admissible, should be persisted in is obvious, not only because the maintenance of life up to the extreme limits which the progress of the diseases admits of depends thereon —and it is our recognised duty as physicians to sustain life even when it is a hopeless burden— but because (to say nothing of the chance there may be of our diagnosis being in some cases erroneous) there may be, at least in the case of tubercular disease, a prospect, however remote, of ultimate recovery.

AFFECTIONS OF THE ABDOMINAL LYMPHATIC GLANDS.

By John Syer Bristowe, M.D., F.R.C.P.

The lymphatic and lacteal glands of the abdomen are frequently the seat of disease; sometimes they become inflamed, sometimes hypertrophied, sometimes tubercular, and sometimes the seat of the various forms of cancerous growth and of degenerative changes.

In inflammation they become enlarged, congested, softened, and tender, and sometimes undergo suppuration, and may then discharge their contents by various routes, and even by rupture into the peritoneum. When the inflammation subsides they may according to circumstances recover their healthy state, or remain enlarged, or become atrophied and indurated. The symptoms indicative of their inflammation are more or less pain and tenderness in the situation of the affected glands, with perhaps hardness or distinct tumour, and more or less violent inflammatory fever. Inflammation of the abdominal glands is probably of very common occurrence, as secondary to inflammation or ulceration of the various organs with which they are in connexion; but we are chiefly acquainted with inflammation of the mesenteric glands in enteric fever, and in dysentery, and of the lumbar glands and those about the brim of the pelvis in connexion with inflammatory affections of the genito-urinary organs.

Hypertrophy of the glands is not very easy to separate from tubercular disease of the glands on the one hand, and from some forms of malignant disease on the other. It is indicated by a more or less gradual increase in their size, attended with a more or less fleshy consistence, and a colour varying between a dull white or buff, and a reddish fleshy hue. It is an affection rarely limited to the glands of a particular part; and generally, therefore, when the abdominal glands are hypertrophied the lymphatics of other parts of the body are hypertrophied also. The symptoms which attend this affection are rarely connected specially with the abdomen; excepting in so far as there may be a tumour there, and more or less impairment of nutrition; they are for the most part those of gradually increasing anæmia, and a form of cachexia, in which sometimes there is a remarkable increase of white corpuscles in the blood (Leucocythæmia).

Tubercular deposits, in the mesenteric glands especially, are not uncommonly associated with similar deposits in the peritoneum and intestines; and they generally form well-defined cheesy lumps embedded in enlarged and more or less congested gland substance. Not very infrequently such deposits take place in glands which have previously undergone hypertrophy, and to such an extent sometimes that whole glands become caseous. Tubercular glands sometimes soften or suppurate and form vomicæ; and very frequently indeed dry up and contract and become converted into inert cretaceous masses. This condition of glands is probably attended with no symptoms distinguishable from the symptoms due to the associated tubercular affection of other abdominal organs which is generally present.

Cancerous disease of the various abdominal glands is common in all its varieties. It is sometimes primary (in which case it is probably generally if not always some variety of what Virchow terms lymphoma). It is more frequently secondary to cancer of other parts; and then, for the most part, the glands chiefly affected are those which are in relation with the organ primarily affected. Thus, in cancer of the testis the lumbar glands become cancerous; in cancer affecting the remaining genito-urinary organs, and other organs situated in the pelvis, the glands which become specially implicated are those in the pelvis, and about its brim; in cancer of the bowels, the mesenteric glands chiefly suffer; and in cancer of the stomach, kidneys, and neighbouring parts, the retro-peritoneal glands of the upper part of the abdomen. Cancerous glandular tumours sometimes attain an enormous size; and it is not infrequently by their growth and disintegration that perforation or obstruction of viscera, and other serious complications, which have been elsewhere sufficiently described, are produced. It is difficult, and would be useless, to discuss the symptoms and effects of such tumours apart from those of cancer of the peritoneum and other abdominal organs, which have been already fully considered.

In addition to the degenerations which follow upon inflammation, and upon the deposition of tubercle, it may be stated that in extreme cases of lardaceous disease, the abdominal lacteal and lymphatic glands may share with other parts in this form of degeneration.

ASCITES.

By John Syer Bristowe, M.D., F.R.C.P.

Pathology.—The accumulation of fluid of a more or less serous character within the peritoneal cavity is called "Ascites," or "Abdominal Dropsy." It is an accompaniment or sequela of numerous different forms of disease; but depends immediately on some condition which modifies the action of the capillary vessels, and in some cases perhaps of the lymphatics, of the peritoneal membrane. This condition may be, in the first place, some morbid process going on in the peritoneal tissue, and affecting directly its minute vessels; or, in the second place, some impediment to the return of blood from them existing in the course of the portal system; or, in the last place, some impediment to the return of blood from them connected with some disease affecting generally the movement of blood in the systemic veins. Among the first of these classes may be included peritonitis, peritoneal tuberculosis, and peritoneal cancer; among the second, tumours or other growths obstructing the trunk or main branches of the vena portæ, chronic congestion and induration of the liver, lardaceous disease of that organ, and especially cirrhosis; and among the last, heart disease, Bright's disease, some affections of the lungs, and perhaps some forms of anæmia.

(1) Acute peritonitis, like acute inflammation of other serous membranes, is doubtless attended in most cases with more or less effusion of serum; but the effusion is rarely abundant and rarely amounts to what would be recognised during life as Ascites. Not very infrequently, however, when the acute peritonitic symptoms have subsided, and the patient appears to be convalescent or even well, abdominal dropsy slowly supervenes. Ascites is especially apt to occur in women in whom the peritoneal inflammation has been connected with some inflammatory condition of the pelvic organs. It is frequently associated with the growth of cystic ovarian tumours; and is then in some cases due either to the occasional rupture of small superficial cysts, or to the establishment of more extensive communications between the cavities of the ovary and that of the peritoneum, and the discharge of fluid from the thus exposed secreting surfaces into the abdominal cavity. In all these cases the peritonitis assumes a su acute or chronic character. Tubercular deposits in connexion wi the peritoneal surface are another fruitful cause of Ascites.

12 out of the 48 cases of tubercular peritonitis analysed on a former page, this condition was present, and several of them had been tapped. Abdominal cancer, again, is frequently attended with dropsical effusion. Of the 22 cases of peritoneal cancer previously considered, 11 had Ascites in a greater or less degree; and it may be added, that dropsy not infrequently attends cancerous disease of the ovaries and other pelvic organs, and of the mesenteric or retro-peritoneal glands. In what degree Ascites, dependent on disease of the peritoneal membrane, may be due severally or collectively to direct involvement of the capillaries and minute veins of that membrane, to obstruction of the lymphatic orifices which seem now proved to exist there, or to increased functional activity on the part of the epithelial cells, is not very easy, perhaps, to decide ; but there is probably little doubt that, in some cases in which there is infiltration and contraction of the peritoneal folds, especially of the mesentery, the larger veins contained within them become, as Oppolzer suggests, obstructed, and that the Ascites is produced or augmented by this obstruction.

(2) Impediment to the passage of blood along the portal vessels, with consequent Ascites, may be caused by various morbid conditions ; occasionally by the pressure on the vena portæ of an aneurismal, hydatid, or cancerous tumour, originating externally to the liver ; more frequently by the pressure of cancerous, "knotty," syphilitic or hydatid tumours developed in the hepatic substance, and especially by cancerous and fibroid growths occupying the lesser omentum, and extending thence into the liver along the capsule of Glisson ; but most commonly by some general hepatic disease which involves the hepatic capillaries and the minute veins which open into and emerge from them. Of the diseases last referred to, cirrhosis is the most frequent and the most important. Cirrhosis, however, though doubtless tending in all cases ultimately to cause Ascites, is sometimes fatal by hæmatemesis before any dropsical effusion has taken place, and is not infrequently found to be present, unsuspected, in death from other visceral diseases. Out of forty-six cases in which cirrhosis was discovered *post mortem*, in twenty only was there more or less accumulation of ascitic fluid. The presence of a fibroid capsule, surrounding the liver, compressing it, and squeezing it into a comparatively small rounded mass, produces the same effect. This formation, which is probably of inflammatory origin, is sometimes associated with cirrhosis, or other morbid states of the liver, but is sometimes present when the liver seems otherwise perfectly healthy, and where it is the sole visible pathological phenomenon associated with Ascites. There is no doubt that lardaceous disease of the liver also sometimes leads to abdominal effusion, and not improbably an extreme state of fatty deposition may have the same result ; but in both of these cases the hepatic affection is almost always associated with still more serious disease in other organs, which is itself capable of causing dropsy, so that the influence of the liver in its causation is rendered some-

what difficult of identification. Similarly, it is quite certain that chronic induration and congestion of the liver, and especially that condition of the organ to which the name "nutmeg liver" is applied, are frequently instrumental in the production of Ascites, although they are themselves always secondary to dropsy-producing diseases, such as kidney disease, heart disease, chronic bronchitis and chronic phthisis.

(3) All the diseases which have just been enumerated, viz. chronic bronchitis and phthisis, heart diseases, and certain forms of kidney disease, which cause anasarca, cause naturally, as a part of that anasarca, effusion of serum into the abdominal cavity: but in most cases the abdominal effusion is proportional only to the effusion in other parts, and fails to be recognised as Ascites. In some cases, however, the dropsical accumulation in the abdomen becomes excessive, while that elsewhere undergoes but little increase. When this happens, it is usually in connexion with, and then probably immediately dependent on, some abdominal complication of the primary disease, especially a congested or indurated, or nutmeg, or even a cirrhosed condition of the liver, or chronic inflammation of the general peritoneal surface, or of that of the liver. But sometimes, even where the ascitic fluid has been so abundant as to need removal by operation, no trace of disease in any of the abdominal tissues or viscera can be discovered. There can be little doubt that in some forms of cachexia and anæmia, in which without there being any apparent visceral disease anasarca takes place, Ascites also occasionally ensues. Yet it may be remarked, that as cases of this kind usually get well, it must generally remain a matter of uncertainty as to whether or not there may have been some slight inflammatory affection of the peritoneum, or some other evanescent local morbid condition on which the Ascites may have depended.

It may be added here, that in a very large proportion of cases of Ascites, several or even many organs are diseased at the same time, so that it becomes difficult or impossible to determine upon what exactly the ascitic accumulation depends. Thus fibroid and lardaceous and other degenerations often affect simultaneously many organs, so that together with the liver we often find the kidneys, the spleen, the lungs, the heart, the blood-vessels diseased in various degrees. Besides which, in all such cases there is a great tendency to inflammatory implication of the peritoneum as well as of other serous membranes; and tuberculosis is often present. This simultaneous affection of many different organs and tissues is specially common among those who have passed a life of debauchery, among those who have laboured under the syphilitic cachexia, and among those who have suffered long from bone-disease, from protracted suppuration, or from chronic tuberculosis.

The amount of fluid present in Ascites may vary from a few pints up to four or five gallons, and indeed much larger quantities are recorded as having been met with. The fluid itself is for the

most part slightly viscid, transparent, of a yellowish or greenish tinge, alkaline and containing both albumen and fibrine (or fibrinogen). It may, however, under different circumstances, become very viscid, opaline, or opaque from inflammatory products, or it may contain blood.

It would be tedious and, it is feared, useless to go at any length into the statistics of Ascites; for in the first place Ascites is an incident only of many different forms of disease, the statistics of which, with those of their particular relations to abdominal dropsy, are all elsewhere sufficiently discussed: and, in the second place, to bring together the statistics of Ascites in the gross, would be to combine a number of heterogeneous figures the manipulation of which could for the most part only lead to useless or fallacious results. There are a few facts, however, which the statistics of a general hospital have supplied me with, which it may be worth while to state. According to these statistics, there is little difference between males and females as regards their respective degrees of liability to Ascites, although undoubtedly hepatic dropsy is far more common in men than in women; Ascites is most frequent in the decades from thirty to forty and from forty to fifty, next in those from twenty to thirty and from fifty to sixty; but it is not uncommon, both above the latter age and in young children; it occurs with about equal frequency as the result of hepatic disease, heart disease, and kidney disease (in the latter two cases, however, generally combined with a congested or nutmeg or contracted condition of liver); it is from about one-half to one-third as common as a consequence of peritoneal cancer, peritoneal tubercle, bronchitis and phthisis severally; and, again, occurs in association with lardaceous disease of organs and ovarian cystic tumours respectively about half as frequently as in connexion with each of the immediately foregoing diseases.

The prospect of the duration of Ascites, and of eventual recovery or death, necessarily depends almost entirely upon the nature of the disease on which the dropsy depends. Now, most of the diseases causing abdominal dropsy are from their nature lethal, and generally, therefore, Ascites must be regarded as a symptom terminable only with death. Yet even in some of these cases it is of very protracted duration, and relief may be afforded several times by tapping before the arrival of the fatal issue. But in some cases, and even when the disease causing it is usually a progressive disease, in chronic peritonitis, in cirrhosis, in the encapsuled state of liver, and probably also in tubercular peritonitis, the dropsy may be sometimes arrested in its progress, or even, temporarily at least, recovered from. In some cases indeed, both in children and in adults, recovery from Ascites (the cause of which thus necessarily remains more or less obscure) is permanent.

SYMPTOMS.—The symptoms due to Ascites alone are very simple and very characteristic of the affection. The accumulation of fluid within

the abdominal cavity causes the abdomen to enlarge and become tense and then sooner or later compresses and obstructs the intra-abdominal veins, especially those connected with the lower extremities, impedes the movements of the diaphragm, inducing difficulty of breathing, and interferes more or less injuriously with the healthy action of the abdominal viscera. It modifies also the patient's gait, making him walk like a pregnant woman, with his legs wide apart, and his head and shoulders thrown back.

The presence of fluid in the peritoneal cavity is generally easy of detection. The abdomen becomes large, uniformly rounded, but with a tendency to spread or bulge in the flanks as the patient lies on his back, tense and more or less smooth and shining, often presenting distended superficial veins and the linear lacerations of the deeper tissues of the skin which are so common in pregnancy. The stomach and intestines being lighter than the fluid, tend to float on its upper surface; and hence generally the highest part of the abdomen, according to the patient's position, is resonant, while the more dependent parts are dull, the line of demarcation between them being for the most part well-defined and horizontal: hence, too, as the patient changes his position, the fluid and the floating bowels, and necessarily therefore the areas of resonance and dulness, change their positions relatively to the abdominal parietes. It may be added that the liver, which is generally if not always of higher specific gravity than dropsical fluid, retreats sometimes distinctly, as the patient lies on his back, from the anterior surface of the abdomen, a stratum of fluid with sometimes a loop of floating bowel occupying the interval. The presence of fluid is further and very importantly indicated by the peculiar thrill which is experienced by the hand laid flat on the abdomen when a ripple or wave is produced in the ascitic fluid by a slight tap or fillip applied to some other part of the abdominal surface. These signs, however, are not always all present, or at least easy to recognise: and not infrequently tumours and other forms of disease simulate or mask abdominal dropsy. Thus when the ascitic fluid is in small quantity and occupies probably the pelvis only, the presence of dulness will scarcely be detected in any ordinary position which the patient may assume: it may generally, however, be certainly recognised if he be made to rest upon his elbows and knees so as to allow the fluid to gravitate to the neighbourhood of the umbilicus. Thus, again, when peritoneal adhesions are present, both the evidence derivable from the relative positions of resonance and dulness, and the variability of these positions, and that also derivable from fluctuation, may wholly fail us. Thus too when the abdomen is enormously distended, the attachment of the stomach and intestines may be too short to allow of any of these parts reaching the surface of the abdomen, and the dulness may be universal: a condition which does not indeed throw any difficulty in the way of ascertaining the existence of fluid, but may make it not quite easy to determine whether the fluid is free in the abdominal

cavity or whether it is contained in a large ovarian cyst. It need scarcely be said that, independently of the evidence afforded by the history of the case, by the form of the abdomen, and by vaginal examination, there is always in ovarian dropsy (unless indeed it be associated with Ascites) resonance in one or other or both flanks in consequence of the position which the tumour always takes in relation to the bowels; yet to insure accuracy it must not be forgotten that even in Ascites there may be a line of resonance in either flank due to the presence there of the colon. It must be added that œdema of the abdominal walls, or fat in them or in the mesentery, or the presence of diffused peritoneal cancer, are often serious impediments to the accurate diagnosis of moderate dropsical accumulations.

In most cases peritoneal dropsy causes merely that uniform distension of the abdomen which has been above described; but the distending force naturally exerts its most marked influence on those parts of the parietes which are weakest; and hence hernial sacs become often very greatly dilated and attenuated, especially perhaps the sacs of umbilical herniæ; hence, too, in some cases of Ascites in females the recto-vaginal pouch becomes greatly distended, and even protruded through the vulva in the form of a tumour, carrying with it as a covering the posterior wall of the vagina. I recollect one case in which the formation of such a tumour caused not only prolapse of the whole of the posterior wall of the vagina, but also of the upper part of the anterior wall together with the os uteri, which latter was found on the convexity of the tumour. The body of the uterus retained its normal position, but its neck had by the traction exerted on it by the gradual descent of the posterior wall of the vagina been attenuated and drawn out to a length of three or four inches. Occasionally Ascites has been relieved by the spontaneous rupture or perforation of some thinned portions of the abdominal parietes.

Œdema of the lower extremities and intervening parts is a very general and early accompaniment of abdominal dropsy. Sometimes it occurs at so early a period as to be the first symptom of disease which the patient himself recognises, and indeed it is not very uncommon for ascitic patients to assert that their illness began with swelling of the legs. There is no doubt that in dropsy from abdominal disease this complication is due to the impediment to the return of blood produced by the pressure of the ascitic fluid on the iliac veins. It increases for the most part with the increase of the conditions on which it depends; and may become as excessive as that from cardiac or renal disease; but it rarely extends beyond the part with which the mechanically-impeded veins are immediately connected, and never becomes general. It need scarcely, however, be said, that when Ascites is connected with diseases of the heart, lungs, or kidneys, general anasarca is very often present. Anasarca due to abdominal dropsy is generally equal in the two lower limbs; and in this respect differs for the most part from anasarca in the legs

resulting from abdominal tumours or from obstruction by clot of the iliac veins.

Shortness of breath is an early symptom, and it increases with the increase of the dropsy. It is not always noticed by the patient himself while he remains quiet in the sitting or semi-recumbent posture. But even at such times the physician will probably observe that the respiratory acts are unduly quick and shallow. Ultimately, however, this symptom becomes very painful and distressing. It is obviously caused by the encroachment of the enlarging abdomen upon the thoracic cavity, by which the diaphragm becomes pushed up, and prevented from performing the movements necessary for perfect respiration. The lower portions of the lungs become consequently more or less empty of air and collapsed; and as might be anticipated from a knowledge of its cause, it is always much aggravated when the patient lies down.

Although in the earlier stages there may be little or no abdominal discomfort, there generally arises in the course of the affection a good deal of aching, which is usually complained of most in the flanks and across the epigastric or umbilical regions. This is probably due to the pressure which the fluid exerts on the various tissues, but more particularly to that which it exerts on the hollow viscera. This pain is sometimes associated with that of distinct colic, and not very infrequently, when the abdomen has become very largely distended, with pain of a peritonitic character. Indeed acute or sub-acute peritonitis is far from rare in the latter stages of Ascites. It may be added, that diarrhœa is not uncommon in the course of Ascites, and that it seems to be sometimes due to the same impediment to the portal circulation which causes the Ascites itself, and is sometimes dependent on some slight dysenteric inflammation; and that although early in the affection there may be no visible morbid condition of tongue and neither thirst nor loss of appetite, the tongue and the digestive functions after a while all become variously and more or less seriously affected. It may be added further, that patients almost invariably complain of flatulency, a complaint which is undoubtedly due in many cases to excessive flatulent distension of the bowels, but may in some degree be explained by the discomfort which, in the presence of much ascitic fluid, even a normal amount of gaseous distension may occasion. There is generally some dryness of skin and some diminution in the urinary secretion.

There are many symptoms more or less grave, besides those which have been considered, which may be presented by ascitic patients; but they are symptoms for the most part due to the diseases upon which the Ascites itself depends, and are sufficiently considered elsewhere under the heads of those diseases.

TREATMENT.—The treatment of Ascites, in a large proportion of cases, merges in the treatment of the disease by which it has been caused. Still, in some cases from the very beginning, and in most when the accumula-

tion becomes very great, special treatment directed against the Ascites is, or appears to be, called for. To promote the absorption and removal of the ascitic fluid there are good theoretical reasons for the employment of those remedial measures which increase the discharges from the skin, the kidneys, and the bowels. The skin in cases of Ascites is usually unnaturally dry, and this fact seems to furnish an additional argument in favour of the use of diaphoretics. There is no doubt, indeed, that diaphoretic remedies are very generally beneficial to the patient. And amongst these must not be forgotten the most powerful of all, namely the hot-bath, the vapour-bath, and the Turkish bath. Again, the frequent diminution in the urinary secretion may be urged as a further motive for the employment of diuretics; and again, it may be stated generally that the promotion of the flow of urine is serviceable. Still more, the close connexion between the peritoneal membrane and the mucous lining of the bowels, and the fact that in hepatic obstructions the mesenteric capillaries sometimes relieve themselves by discharge of serum at the serous surface, sometimes by the escape of serum or blood at the mucous surface, would seem to be decisive as to the value of purgatives, and more especially of watery purgatives; and it may be freely admitted that purgatives are very often beneficial. I must confess, however, that although fully acquiescing in the importance of restoring as far as may be, and of maintaining, the healthy action of the skin and kidneys, and of promoting a tolerably free action of the bowels, I have never, to the best of my recollection and belief, seen an ascitic patient materially relieved as regards his Ascites, far less cured, by a course of either diaphoretics, diuretics, or purgatives. And in respect to purgatives I may add, that I have frequently had to discard them because, while they were not distinctly benefiting the dropsy, they were obviously affecting the patient's health injuriously; and further, that according to my own experience diarrhœa is a not infrequent concomitant of Ascites, and is often difficult to arrest, and often of bad augury. There are, however, certain medicines which are more or less diuretic in their action which have been, or are, supposed to have, occasionally at least, a specific influence over dropsical accumulations in serous membranes, and under the use of which occasional recoveries are recorded. Among these may be enumerated mercury, iodide and bromide of potassium, copaiba, and the combination of fresh squills and crude mercury.

But it must be repeated that, as a rule, the treatment which is directed towards the alleviation or cure of the disease or condition of health to which the Ascites is secondary, is that which is most likely to be curative as regards the dropsy. The modes of treating heart diseases, kidney diseases, bronchitis, cirrhosis, and so on, need not be here discussed; but it may be pointed out that in a considerable number of cases of Ascites, and even in many of those in which the Ascites is dependent on the diseases which have just been enumerated, there is present a greater or less degree of anæmia and want of tone,

and that in some at least of these cases anæmia and want of tone are in some degree instrumental in producing the dropsy. It is certain that tonics are very often well borne by ascitic patients, and that even when not well borne at first a little judiciousness in their employment, or in the employment of other preparatory measures, will render them tolerable; and it is certain that under their use ascitic patients do often not only improve in health, but lose, in part or wholly, their dropsical accumulation, and that occasionally the recovery is permanent, and permanent even after the performance of paracentesis. Quinine, iron, and cod-liver oil are probably the most valuable forms of tonics.

Counter-irritants and other forms of local applications are doubtless sometimes useful for the relief of uneasiness and pain; but no such applications are of use in promoting absorption of the fluid. But when the abdomen has become very much distended, and the patient is suffering seriously from the inconvenience and distress which attend such distension, the removal of the dropsical fluid by paracentesis becomes necessary. The time for the performance of this operation must be determined for each case, less by the actual distension of the belly than by the gravity of the symptoms which attend that distension. The operation is generally postponed as long as possible, and I believe rightly; but it may be worth while to state that it has not very infrequently appeared to me that the beneficial effects of remedies have been exerted in a much greater degree after paracentesis than while the belly was largely distended. Paracentesis is generally a harmless operation; but sometimes peritonitis ensues, and is apt to be rapidly fatal. I believe that in cases of peritoneal dropsy dependent on cancerous disease of the abdomen tapping is not only very rarely of even temporary benefit, but that it generally hastens death. Iodine and other substances have occasionally been injected into the peritoneum for the cure of Ascites, and successful cases of this hazardous kind of treatment are recorded.

§ II.—DISEASES OF THE DIGESTIVE SYSTEM (*continued*).

F.—DISEASES OF THE LIVER.

1. HEPATALGIA.

2. CONGESTION OF THE LIVER.

3. JAUNDICE.

4. BILIARY CALCULI.

5. SUPPURATIVE INFLAMMATION
    OF THE LIVER; HEPATITIS.

6. CHRONIC ATROPHY OF THE
    LIVER; CIRRHOSIS.

7. ACUTE ATROPHY OF THE
    LIVER; YELLOW ATROPHY.

8. FATTY LIVER.

9. CARCINOMA.

10. HYDATIDS OF LIVER.

11. WAXY LIVER.

HEPATALGIA.

By Francis Edmund Anstie, M.D.

Definition.—An affection characterised by attacks of deeply seated pain in the region of the liver; intermittent, in the manner of neuralgias; attended by no organic changes or febrile disturbance; not necessarily involving any interference with secretion; but occasionally attended with arrest or perversion of the biliary secretion and consequent jaundice and stomach-derangement.

Clinical History.—It must be confessed that there are many difficulties in drawing a clear and recognisable picture of this disease, of the separate existence of which I am, however, convinced. Some of these difficulties will be more fully pointed out under the head of "Diagnosis;" but it will at once be evident that the deep situation of the liver within the body must render it far from easy to localize so impalpable a thing as pain with certainty in the hepatic nerves. Valleix, indeed, does not hesitate to throw serious doubt on the identification of Hepatalgia as a substantive affection, and his great authority has probably induced many to discredit its existence as a separate disease. Nevertheless, the following picture, drawn from my experience of cases which have been under my own notice, will probably be thought to represent a sufficiently distinctive malady.

The subjects of Hepatalgia are probably never troubled *only* by pain in the liver; they are persons of a nervous temperament, in whom a slight shock to, or fatigue of, the nervous system habitually provokes neuralgic attacks; the pain localizing itself sometimes in the branches of the trigeminal, sometimes in those of the sciatic, sometimes in the intercostal nerves, &c. In one instance which has been under my observation, the attacks of Hepatalgia alternated with cardiac neuralgia, assuming the type of a rather severe angina pectoris. In another case the patient, a man aged sixty-seven, was very liable to attacks of intermittent abdominal agony, in which one could hardly doubt that the pain was located in the colon, and was attended with paralytic distension of the bowel; the peculiar feature of the case being the sudden way in which the symptoms would appear and depart, independently of any recognisable provocation or the use of any remedies. On two separate occasions this patient was attacked with pain of a precisely similar kind, but limited to the right hypochondrium, attended

with great depression of spirits, and followed by a well-pronounced jaundice. So remarkable was the conjunction of symptoms in these two attacks that a strong suspicion of biliary calculus was raised, but not the slightest confirmation of this idea could be obtained; and indeed one symptom—vomiting—which nearly always attends the *painful* passage of a biliary calculus, was altogether absent.

Putting aside a considerable number of cases in which "pain in the liver" was vaguely complained of by patients who were plainly hypochondriacal, and whose account of their own sufferings could not be relied on, I have altogether seen five instances of what I regard as genuine Hepatalgia. The first of these was very remarkable in its history and in all its features. The patient was a respectable girl of eighteen, subject to migraine, who had reason to fear that she had become pregnant, though this proved, ultimately, not to be the case. Under these circumstances she was attacked with intermittent pains, in the right hypochondrium, of intolerable severity; resembling, in fact, the pain of biliary calculus, but without the sense of abdominal constriction, and without any vomiting. These recurred daily at about the same hour in the morning, for about ten days; when, rather suddenly, a jaundiced tint appeared upon the face, and very shortly the whole skin was coloured bright yellow; there was intense mental apathy; the urine was loaded with bile-pigment, and the fæces clay-coloured. This state of things lasted only about a week, and then very rapidly disappeared; but as the jaundice subsided there was a partial recurrence of the neuralgic pains, which, for a day or two, were as severe as they had ever been. The other four cases of Hepatalgia which I have seen, including that of the man above mentioned, have all been in persons in advanced life; but, except the latter, neither of them displayed any symptoms of disordered biliary secretion; and the diagnosis (as to *situation*, for the *character* of the attacks was manifestly neuralgic) rested mainly on the fact that the pain radiated to the shoulder.

There remains to be noticed one clinical feature of the disease, which, I believe, is characteristic; namely, the peculiar mental depression which attended all the cases I have seen, but was most marked in the two in which jaundice occurred. In the girl above referred to, the apathy, during the period when there was jaundice but no pain, was even alarming; it reminded one of the mental state in commencing catalepsy; during the painful stages it was more like the gloom of suicidal melancholia. Of course, the acute mental anxiety which this patient had suffered would account for a good deal of this; but the symptom was as distinct, though less severe, in the case of an elderly lady, whom I have attended on another occasion for migraine; here there was no recognisable source of anxiety; and, on the other hand, there was no reason to suspect the retention of l elements in the blood. It seems, therefore, as if an essentially depr ing influence on the mind was excited by hepatic neuralgia; or that emotional causes are the chief source of the malady.

PATHOLOGY AND ETIOLOGY.—Of the true pathological conditions of Hepatalgia we know even less than of those which prevail in other neuralgias: the one antecedent fact recognisable is the existence of nervous depression. It is probable that pure neuralgic pain never attacks organs so slenderly provided with sensory nerves as the liver, except in subjects who belong to families of a markedly neurotic type, and that this hereditary constitution is the main predisposing cause. Of exciting causes, the most probable are mental shock or continued anxiety of mind: one may also suppose it probable that the gouty taint might excite Hepatalgia, as it undoubtedly can excite gastralgia; but I have seen no such case.

DIAGNOSIS.—(*a*) The first difficulty in identifying a case of Hepatalgia is the exclusion of painful affections of the abdominal walls. Myalgic affections of the abdominal muscles are very common, but the distinction of these from Hepatalgia may be established with certainty; there will always be a history of undue and persistent strain upon the muscles, from the nature of the patient's occupation, or else from some unusual exertion which he has made; and the pain itself will be diffused, never completely intermitting, and strikingly aggravated by movements. (*b*) Abdominal neuralgia, *i.e.* neuralgia of the abdominal branches of the dorsal and lumbar nerves, is more difficult to distinguish from Hepatalgia; and cases might easily arise in which it would be impossible to say whether the pain was parietal or visceral; unless, indeed, we are warranted in supposing that the mental depression already mentioned as attendant on all the cases of Hepatalgia which I have seen is a truly characteristic and necessary attendant of the latter affection. The absence of "*points douloureux*" would tend to exclude the theory of parietal neuralgia. (*c*) The violent pain which attends the difficult passage of a biliary calculus towards the bowel strongly resembles neuralgia in some cases, though it is usually too continuous and unremitting to be long mistaken for it; but the great points of distinction are the absence, in Hepatalgia, of the sense of constriction, and the vomiting, which almost universally mark the passage of a calculus. (*d*) The existence of malignant disease in the liver or the neighbouring organs might give rise to paroxysms of lancinating pain wholly undistinguishable in themselves from neuralgia; and in the early stages, before the constitutional signs of the diathesis were well marked, it might be impossible to make an accurate diagnosis. (*e*) Abdominal tumours of any kind, but more especially aneurism of the aorta or of the cœliac axis, have been known to cause agonizing pain of an intermittent type, apparently referable to the region of the liver: and in this case nothing but the progress of the disease, and the ultimate development of the physical signs, could establish the diagnosis. (*f*) Accumulation of fæces in the ascending and transverse colon has sometimes given rise to pain closely resembling neuralgia, and referred to a point deep in the right hypochondrium: here the existence of obstinate (though not necessarily

complete) constipation will assist our judgment; and, besides, this kind of pain is usually felt in the right flank, and in the back, as severely as in front.   (*g*) Last, and most difficult of all pains to be distinguished from genuine Hepatalgia, is the severe pain in the right side so often experienced by hypochondriacal patients.   Nothing can avail us here but a thorough and comprehensive review of the whole clinical history of the case, and of the antecedents and family history of the patient; and for this purpose we must refer the reader to the description of the symptoms of hypochondriasis, and of the hypochondriacal constitution generally, which are to be found in the second volume of this System of Medicine.

PROGNOSIS.—There is no reason to think that Hepatalgia ever assumes the type of the inveterate and incurable neuralgias, although the suffering may be extremely severe while it lasts.   It seems rather to be an occasional and transitory affection, occurring in patients liable to neuralgic affections of other parts.   The persistence of pain, otherwise corresponding to the type of Hepatalgia, for any great length of time, should always lead us to suspect that there is, in reality, some permanent organic cause of pressure upon the nerves.

TREATMENT.—I am unable to speak from personal experience of the treatment of Hepatalgia arising from malarious poisoning, if such an affection exists, as is probable enough.   We can hardly doubt that quinine would be the appropriate and most efficacious remedy in such cases.   But in regard to Hepatalgia, as recognised by me in the five cases already referred to, I can state, decidedly, that quinine afforded no relief, while in all of them another remedy *did* effect striking good. The first case that came under my notice was that of the girl who, as already stated, was suffering from severe mental anxiety in consequence of imagining herself to be pregnant: here the tentative use of sal volatile gave some relief, and the *muriate* of ammonia was then administered in half-drachm doses every four hours.   Nothing could be more striking than the amendment in this case.   The drug was administered during the stage of jaundice, and that symptom immediately began to disappear with great rapidity; the neuralgic pains reappeared, but only for a day or two, and in a comparatively mild form; they ceased altogether after a day or two.   In every subsequent case of Hepatalgia my experience was the same; quinine afforded no relief, muriate of ammonia quickly produced improvement, and in a very few days the pain altogether disappeared.

CONGESTION OF THE LIVER.

By W. C. MACLEAN, M.D.

DEFINITION.—Hyperæmia of the gland, characterised by equable enlargement in every direction; a dusky and sometimes jaundiced complexion, with a sense of uneasiness in the right side, not amounting to pain; weight and fulness in the right hypochondrium, apt, if long continued, to be followed by atrophy of the hepatic parenchyma, and by cirrhosis, in men of intemperate habits. Caused by (a) obstruction to the circulation in the chest, (b) by malarial fevers, (c) by the use of too rich food and the abuse of fermented and spirituous liquors, (d) by chills following exertion.

PATHOLOGY.—Whatever interferes with the circulation in the chest indirectly tends to produce hyperæmia of the Liver. Valvular disease of the heart, such as incompetency of the mitral valve, causes stagnation of blood in the vena cava and hepatic veins; in time "this is propagated to the portal vein, and to the organs in which this vessel takes its origin" (Frerichs). The liver becomes turgid with blood, and congestion extends to the venous system of all the digestive organs; the skin, at first dusky from imperfect aëration of the blood, becomes finally jaundiced from impeded elimination of bile. The result of this capillary congestion, if long continued, is diminution in the size of the organ "from atrophy of the hepatic cells" (Budd, Frerichs).

Congestion of the Liver, the result of malarial fevers, has already been described (article Malarial Fevers, Vol. I.). Dr. Graves regarded the hyperæmia of the Liver so often observed in intermittents, as a form of hepatitis. Whatever may have been the case within the sphere of this eminent physician's observation, inflammation of the Liver, in the course of a malarial fever, is an extremely rare occurrence in India. Out of 243 cases of intermittent fever, Morehead observed inflammation of the Liver in six cases only. In remittent fever true hepatitis is equally rare, but Congestion of the Liver is more frequent than in intermittents.

That Congestion of the Liver often follows the undue consumption of stimulating food, and the abuse of fermented liquors and ardent spirits, is generally allowed. I have seldom known a great beer-drinker in India, who was not subject to attacks of Congestion of the

Liver, unless he happened to be a man of very active habits with vigorous respiration. Spirit-drinkers eventually suffer from adhesive inflammation of the connective tissue of the gland, with all its grave consequences, but it is not the less true that in the early days of this baneful habit they are liable to hyperæmia of the gland.

Morehead denies that Congestion of the Liver follows the direct transmission of noxious matters of food by the portal blood from the intestine to the Liver, on the ground that the assimilable portion of the food is absorbed in the upper parts of the canal, and that the fluid part of the residual matters, which is absorbed from the large intestine, consists solely of the innocuous parts of the mass.

If it be true that nature possesses a power of selection to such an extent as this opinion implies, habitual diners-out and lovers of the "good things" of the table ought to suffer less than, I suspect, is common in their experience. I need scarcely add that, although Morehead gives our assimilative organs more credit for judicious selection than I fear they deserve, he only calls in question Dr. Budd's mode of explaining hyperæmia of the Liver from over-indulgence at table, for he freely admits that excess in eating and drinking deranges the functions of the Liver in common with those of the digestive organs.

Congestion of the gland often follows chills after violent exercise. Some of the most marked cases of the affection seen by me in hot climates have followed chills after severe exertion in the racket-court, more particularly when the game has been played too soon after eating.

SYMPTOMS.—Whatever be the cause, enlargement of the organ in all directions is a constant symptom of hyperæmia of the Liver. The gland will be found to extend from an inch to an inch and a half below the level of the ribs, and as much beyond its normal limits in the mammary region. The patient complains of a sense of fulness and weight in the right side, and a change of position from the right to the left side aggravates his uneasy sensations. In that sad class of cases depending on organic disease of the heart, the symptoms, general and local, characteristic of the heart-affection, will be present in proportion to the extent and duration of the organic disease, viz., dyspnœa, dinginess and jaundice of the countenance, dropsical effusion, and the abnormal heart-sounds characteristic of the valvular lesion. Frerichs has noticed that the urine is scanty, dark-coloured, and albuminous in cases depending on obstructed circulation, and in several men dying at Netley from the consequences of disease of the mitral orifice, with enlargement of the Liver and great derangement of all the digestive organs, the same dark, scanty, and albuminous condition of the urine was noticed.

MORBID ANATOMY.—A congested Liver is enlarged in all directions. The capsule is smooth and stretched. The gland bleeds freely

section. In cases of longer standing, less blood follows the knife, but what has been called a nutmeg-like appearance is seen (Bright, Frerichs). Sometimes, as is recorded in the note of the post-mortem examination of one of the patients referred to above, the parenchyma has a uniform dark-red hue "with branching dark streaks marking the positions of the hepatic veins." The other abdominal viscera are usually congested with serous blood.

DIAGNOSIS.—In cases depending on obstruction to the circulation from heart-disease, there need be no difficulty in the diagnosis. When cardiac symptoms are sufficiently developed to cause any appreciable Congestion of the Liver, they declare themselves by their characteristic signs, which can hardly escape the notice of an attentive physician.

The diagnosis, between mere Congestion of the Liver and commencing hepatitis, is important in hot climates. Enlargement of the Liver, with fulness, weight, and uneasiness not amounting to pain, if they occur in a part of the world where suppurative inflammation of the gland is common, will always cause anxiety. If the above symptoms are present in the course of a malarial fever, they need not be regarded with alarm, for while Congestion of the Liver in such diseases is common, inflammatory action leading to suppuration is extremely rare. In passive congestion the patient is tolerant of free palpation and percussion, there is no sympathetic pain in the shoulder, no fever, no increased frequency of the pulse. When pain and enlargement coincide, the case is more serious. A careful physician, bearing in mind the extreme importance of the diagnosis in doubtful cases, will not fail to examine them with the greatest care.

PROGNOSIS.—In congestion depending on mere errors in diet, or occurring in the course of a malarial fever, the prognosis is of course favourable. When hyperæmia of the gland is the result of organic disease of the heart, the unfavourable prognosis is governed almost entirely by considerations connected with that disease.

TREATMENT.—Congestion of the Liver, the result of indulgence at table, is easily remedied, provided the patient co-operates with the physician. The action of a few mild saline purges, abstinence at first, and a more guarded regimen for the future, usually suffice; the surface may be stimulated by a turpentine stupe, and if the kidneys are not acting well, saline diuretics should be given. Beer-drinkers, suffering in this way, should be advised to substitute the light wines of France for their favourite beverage, and to dilute them as the French do. Spirit-drinkers should be warned of the inevitable consequences of their habits. Not many months ago, a gentleman was landed at Southampton from Ceylon, dropsical from cirrhosed liver: my friends Mr. King Sampson and Dr. Trend will remember the case well. This gentleman told me his sad history : " Five years ago I consulted Dr. Budd for Congestion of the Liver, who advised me to change my

way of living, and warned me that if I did not I should die dropsical.
I returned to Ceylon and to my old habits, and here I am."—The
treatment in cases depending on heart-disease must be palliative,
and should be carefully conducted. Where circumstances admit of
it, and the case is not too far advanced, the patient may be advised
to visit a suitable watering-place. Patients will often do this, who
cannot be persuaded to take what is recommended in the shape of
physic. Frerichs recommends the Ragoczy spring of Kissingen, or
the Mill springs of Karlsbad, or the waters of Marienbad. A few
leeches to the anus often give much relief, but cannot be often re-
peated : it is a common remedy in Indian hospitals. Attention
must be paid to the bowels and kidneys, which should be kept active
by a judicious selection of aperients and diuretics. As a congested
state of the Liver adds greatly to the patient's distress, no pains
should be spared to lessen it by such means as are indicated above,
and the patient's diet should be of as unstimulating a quality as
possible.

When permanent enlargement of Liver or spleen, with or without
induration, follows the congestion incident to repeated attacks of
malarial fevers, or long residence in malarial places without such
attacks, immediate change of climate is necessary. In cases of this
kind benefit often follows the prolonged use of a combination of iron
and quinine, such, for example, as Dr. Easton's syrup of the phos-
phates of iron, quinia, and strychnia, a formula for which will be
found in the first volume of this work, p. 65. Great comfort and
support is given by the use of what is called the hydropathic belt,
which keeps up steady pressure on the distended parts.[1] The more
obstinate cases rarely resist the application of the ointment of the
biniodide of mercury, used in the manner described by me in the
article on Intermittent Fever, in the first volume of this work, and in
the Medical and Statistical Reports of the Army.

A more extended use of this ointment at Netley, in this class of
cases, has shown this to be a remedy of real efficacy.

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[1] These belts are made of stout calico shaped to the abdomen with cross pieces of tape
on the inner side, which keep next to the skin a fold of cloth wrung out of cold water,
and a piece of waterproof cloth or oiled silk to prevent evaporation.

JAUNDICE.

By Edward Goodeve, M.B.

DEFINITION.—Jaundice, or Icterus, is a condition attending many diseases involving the liver or biliary passages, known by yellowness of the tissues and of many secretions of the body, and associated with symptoms of various degrees of severity affecting the vascular and nervous systems and the functions of digestion and nutrition. It is in reality a secondary affection, the causes of which are numerous and of widely different character, but from its importance it is generally considered worthy of separate notice in works of medicine.

ETIOLOGY.—In Jaundice the discharge of bile into the duodenum through the common duct is wholly or partially prevented, or, according to general but by no means universal opinion, the secretion itself is suppressed. The bile may be mechanically retained in the biliary passages or receptacles, and being absorbed therefrom become diffused through and colour the tissues; on the other hand, there may be no such mechanical impediment and Jaundice may nevertheless exist.

Jaundice may be produced artificially in animals by ligature of the common duct, so that it can be proved experimentally that complete obstruction of the flow of bile into the intestine is adequate to cause the disease. The mechanism of Jaundice from obstruction is easily understood, but, as before said, Jaundice occurs also in cases in which no obstruction can be discovered, as when it follows mental emotion, certain cases of blood-poisoning, many fevers, pyæmia, poisoning by phosphorus, snake-bites, &c. These cases are much more difficult of explanation, and their causation has given rise to many theories and to great differences of opinion.

The following are among the principal views that have been held as to the cause of the non-mechanical forms of Jaundice :—

1. That the bile, not being eliminated or excreted by the liver, accumulates in the blood and dyes the tissues yellow. This attributes the Jaundice to suppression of secretion.

2. That in some diseases the hæmatin of the blood is changed into bile-pigment with disintegration of a large number of blood-corpuscles, assigning a blood-origin to the colouring matter of Jaundice.

3. The opinion of Professor Frerichs, who attributes non-obstructive Jaundice either to abnormal diffusion of bile, arising from some alteration in the circulation of the blood in the liver, or else to defective metamorphosis, or impaired consumption of bile in the blood. His views are stated in the two following propositions :—

1. Increased absorption of bile from the liver into the blood.

2. Through small consumption or sparing change of the bile which has been taken up into the blood.[1]

These three views demand a brief consideration :—

The first or suppression theory supposes that bile exists preformed in the blood, and that it is merely filtered off by the liver, not formed in it ; and that owing to some derangement, structural or functional, of that organ, it accumulates in the blood as urea does in certain diseases of the kidney, or when they are extirpated in animals.

Bile is a compound fluid containing colouring matter, cholepyrrhin or biliverdine, and two acids, glycocholic and taurocholic, the acids combined with soda-bases. It is possible that some of the constituents of this compound liquid may be formed in the liver, and that others are merely separated by it from the blood. Most physiologists admit that the bile-acids are formed in the organ, but there is more difference of opinion about the pigment, and it is in the behaviour of this constituent in Jaundice that the chief interest centres. It is supposed by some to exist in minute quantities in the blood, and many writers believe that it is merely separated therefrom by the liver. This view of suppression of secretion of the colouring matter in Jaundice is supported by Dr. Budd[2] and by Dr. Harley,[3] who admit that the biliary acids do not pre-exist in the blood. The most serious objection to the opinion lies in the fact that the colouring matter does not accumulate in their blood when the liver in animals is extirpated.

The second view is founded on the supposed intimate relations between hæmatin and bile-pigment. Hæmatin has not actually been converted into bile-pigment in the laboratory, but the changes in the blood extravasated in contusions, and the yellow sputum in some cases of pneumonia without Jaundice, the yellowness of newly-born infants, show the probability of a connexion between them. It has been argued by Breschet, Virchow,[4] and others, that in some diseases the transformation takes place in the blood-vessels, and that thus a supply of yellow pigment is created independently of the action of the liver, constituting a Jaundice of blood-origin. Virchow has lately modified his views as to the frequency of the occurrence of Jaundice of blood-origin, believing that many of the cases which

[1] Frerichs, Clinical Treatise on Diseases of the Liver, Sydenham Society's Translation, vol. i. p. 85.

[2] Diseases of the Liver, second edition, p. 458.

[3] Harley on Jaundice, p. 22.　　　　　　　　　　　[4] Virchow's Archiv, vol. i.

he formerly held to be due to blood-changes are caused by a catarrhal state of the passages.[1]

The third view, that of Frerichs, contains, as already stated, two propositions. In the first, that, owing to deranged hepatic circulation the bile may be supposed to tend towards the veins instead of towards the bile-ducts; that, for instance, this would be facilitated by circumstances which would diminish tension in the portal vein. In the second case, that the biliary acids, which are normally absorbed into the blood and there undergo changes, probably of oxidation, which end in the production of urinary pigment, have their progress interfered with in fevers and the other abnormal states already mentioned; the metamorphosis ending in the stage of bile-pigment which circulates with the blood and is diffused through and colours the tissues. According to the first proposition, the liver would play an important part, but less so in the second.

It is impossible in this place to give a detailed account of Frerichs' reasons for these views. They will be found in the New Sydenham Society's translation of his work. Neither will space permit any detailed examination of the other opinions.

Frerichs' views have given rise to much discussion and differences as to matters of fact. Thus, Frerichs denies that the bile-acids appear in the urine in any case of Jaundice; they should do this if they circulated unchanged in the blood; and in this he is supported by Stadeler, Neukom, and others. On the other hand, Kühne, Harley, and others assert that they are to be found in the urine in cases of obstructive Jaundice. Frerichs denies also that the bile-acids are to be found in the blood at any time. Kühne's experiments tend to show that they can be detected in the blood in about twenty-four hours after ligature of the common duct in the dog.[2] Dr. Harley believes he can reconcile these opposite statements as regards the urine. He thinks that the discrepancy depends upon the nature of the cases observed, and asserts unhesitatingly that in Jaundice with obstruction the bile-acids will be found in the urine as long as the secreting tissue of the liver continues to act efficiently, but that in cases without obstruction, with complete suppression, they are not to be found.[3] This is consistent with the theory of bile being composed of a filtrate and a secretion. He points out that, even in cases of obstructive Jaundice, the bile-acids may be absent from the urine in the later stages, because in the progress of the disease the secreting tissue is destroyed, and thus that suppression naturally succeeds permanent obstruction. Dr. Harley considers that, the majority of cases of Jaundice being non-obstructive, the acids would be frequently absent in the urine. It is possible, therefore, that a want of discrimination of the nature of the cases examined has led to the belief in some writers of the constant absence of the bile-acids in the urine during Jaundice.

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[1] Virchow's Archiv, vol. xxxii. part i.   [2] Beale's Archives, vol. i. p. 350.
[3] Harley on Jaundice, pp. 59, 60.

Dr. Murchison supports Frerichs' views. He believes that a great portion of all the constituents of the bile are normally absorbed into the blood from the intestinal canal and are at once transformed, so that neither bile-acids nor bile-pigment can be discovered in the blood, and there is no Jaundice; but that in certain morbid states the absorbed bile does not undergo the normal metamorphosis, circulates with the blood, and colours the tissues. He infers that the only difference between Jaundice with obstruction and Jaundice independent of obstruction of the common bile-duct is that, in the former case, none of the bile can escape from the body by the fæces, and, consequently, all that is secreted after the gall-bladder and biliary passages are fully distended must be absorbed into the blood in quantities beyond its power of metamorphosis.[1]

Frerichs' theories depend upon the supposed absorption of bile from the liver and intestines; but it must be remembered, however probable this may be, that there is no positive proof of it, or that the biliary matters are not decomposed in the intestine.

It is impossible within the limits of this paper, nor would it be profitable in the present state of our knowledge, to enter into a detailed examination of the different opinions held upon the causation of non-obstructive Jaundice, with regard to which both observation and experiment have given such contradictory and irreconcilable results. The whole subject requires further investigation before safe conclusions can be drawn.

It seems reasonable to suppose, however, that the secretion of the liver really is diminished in certain stages of non-obstructive cases. In many instances post-mortem examinations show a pale or clear state of secretion and an empty state of the ducts, which would imply that the secretion had been diminished before death; but it does not necessarily follow that, in the initial stages, suppression of secretion had occurred, and that this stood in the relation of cause to the Jaundice.

Whatever view we may take as to the causation of the yellowness of the skin, Jaundice is, in practice, divisible into two classes, as below. The greater number of cases of Jaundice are found to be in the second class.

A. Those dependent on obstruction. Retention of bile, wholly or partially, with absorption.

B. Those without obstruction. In these, according to many opinions, the Jaundice is due to suppression of secretion, owing to functional or structural disease of the liver; according to others, to insufficient metamorphosis of bile, or some change in the blood itself. There may be a mixture of these two classes, the first form merging into the second.

A. Obstructive Jaundice may depend on—
1. Catarrhal conditions, or thickening or swelling of the lining membrane of the gall-ducts, or of the mucous membrane of the duodenum, involving the mouth of the common duct.

[1] Murchison, Clinical Lectures on Diseases of the Liver, pp. 312, 313.

2. Narrowing or occlusion of the ducts by permanent thickening of the lining membrane, or adhesions or cicatrizations of ulcers of the duodenum.

3. Compression of the cauals from without by abnormal tissue.

4. Cancerous, hydatid, aneurismal, or other tumours, enlarged glands, abscesses of liver or pancreas, growths or enlargements of kidneys or· ovaries or other parts, placed so as to press upon the common or other ducts.

5. Masses of fæces in the colon pressing upon the duct.

6. Pregnancy in its later stages.

7. Lumbrici, hydatids, or foreign bodies entering the common duct, and becoming impacted in the passage.

8. Gall-stones blocking up the common duct or secondary canals.

9. Congenital deficiency of bile-ducts.

B. Non-obstructive Jaundice is met with under the following conditions :—

1. Lesions of innervation.

2. Congestions of the liver, active and passive.

3. Malarious fevers.

4. Relapsing and other fevers.

5. Pyæmia.

6. Poisoning by snake-bites, phosphorus, chloroform, &c.

7. Acute and chronic atrophy.

8. Diseases of the portal vein.

9. Excessive secretion of bile with reabsorption.

It is quite possible that in some of the forms of non-obstructive Jaundice, as in the forms with congestion of the liver, malarious and relapsing fevers, the cause may really be mechanical. A swollen and enlarged liver, by narrowing of the ducts or by altering their direction, may cause delay in the onward course of the bile, and permit of absorption and Jaundice. As before mentioned, Virchow[1] has lately advanced the opinion, that a catarrhal or swollen state of the main duct or plugs of mucus are the causes of Jaundice in many cases in which he had formerly attributed it to blood-changes.

Jaundice occasionally occurs epidemically. In these cases it is probably accompanied with congestion of the liver, and in some cases with catarrhal states of the duodenum, mostly due to some form of blood-poisoning.

The most recent classifications of the causes of Jaundice are given by Dr. Harley[2] and Dr. Murchison.[3] As they are exponents of opposite opinions, they are given below.

According to Dr. Harley's outline, they are—

CLASS A.—Jaundice from Suppression.

1. Innervation.

2. Disordered hepatic circulation.

3. Absence of secreting structure.

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[1] Archives, vol. xxxii.   [2] Harley on Jaundice, p. 20.   [3] Op. cit., p. 316.

CLASS B.
1. Congenital deficiency of bile-ducts.
2. Accidental obstruction of bile-ducts.

Dr. Murchison's outline is as follows :—
CLASS A.—Jaundice from Mechanical Obstruction of the Bile-Duct.
1. Obstruction by foreign bodies within the bile-duct.
2. Obstruction by inflammatory tumefaction of the duodenum, or of the lining membrane of the duct, with exudation into its interior.
3. Obstruction by stricture or obliteration of the duct.
4. Obstruction by tumours closing the orifice of the duct or growing in its interior.
5. Obstruction by pressure on the duct from without.

CLASS B.—Jaundice independent of Mechanical Obstruction of the Bile-Duct.
1. Poisons in the blood interfering with the normal metamorphosis of bile.
2. Impaired or deranged innervation interfering with the normal metamorphosis of bile.
3. Deficient oxygenation of the blood interfering with the normal metamorphosis of bile.
4. Excessive secretion of bile, more of which is absorbed than can undergo the normal metamorphosis.
5. Undue absorption of bile into the blood from habitual constipation.

SYMPTOMATOLOGY AND PATHOLOGY.—It will be well, in the first place, to consider the effects of Jaundice generally, and afterwards briefly to notice the principal forms of the complaint, with the mode of discriminating them.

In the fully-formed stages the signs of Jaundice are evident enough in the yellowness of the external surface. This and most of the secretions are more or less of light yellow, yellow, dark yellow, dusky or greenish, or blackish green, according to the intensity of the Jaundice. The yellowness may become well marked, slowly or rapidly, within a few days or within twenty-four hours; a slight tinge of the conjunctiva or slight yellowness of urine being often discoverable before alterations of the skin; the order of succession of colouring being generally urine, conjunctiva, skin. Few of the tissues or secretions escape being coloured during the progress of the disease, but the mucous membranes and mucous secretions are rarely tinged. Morbid collections of fluid, as dropsies and the serum of blisters, become yellow. The greatest amount of pigment passes off through the kidneys, and after that through the sweat-glands (Frerichs).

The urine in Jaundice changes both in colour and composition. The colour depends on the bile-pigments, and varies from a light bright a saffron yellow to dark greenish brown or porter hue ; the quantity often diminishes in the beginning of the Jaundice, owing to congestion

of the kidneys; and afterwards is generally natural if the progress of the disease is favourable. The urine is acid, and besides bile-pigment may contain bile-acids (?), leucine and tyrosine. There are variations in the quantities of urea and uric acid, and sometimes they are greatly lessened. In the later stages of unfavourable cases sugar may appear (Harley). It seems that benzoic acid taken internally does not appear in the urine as hippuric acid. The urine need not be tested for the sake of diagnosis of the disease in well-marked cases of Jaundice, but in the incipient stages an examination might show that it was coming on. Dr. Harley states that, even when Jaundice is well established, examination of the urine gives valuable assistance in the diagnosis of important attendant conditions. In doubtful cases he says that it may help to decide between obstruction or non-obstruction by telling of the presence or absence of the bile-acids or of leucine or tyrosine, and it may give us information as to the state of nutrition by the variations in the natural constituents of the urine.[1] Dr. Murchison doubts both the chemical and clinical value of Dr. Harley's test for deciding on the presence or absence of bile-acids: chemically, because there is no attempt to separate the bile-pigments before applying the tests; and clinically, because it has failed in his hands.[2]

The chemical tests used for the detection of bile-pigment and bile-acids are as follows:—

For the colouring matter, a piece of white rag or white filtering paper dipped into the urine will have a yellowish tinge after drying, if there is any appreciable quantity of bile in the suspected urine. Nitric acid: if a few drops of nitric acid and of urine are placed separately on a white porcelain saucer or plate, and then held so that the two fluids can run together, a play of colours occurs at the line of junction of the two fluids, changing from violet to green, and ultimately to red. The colours fade away quickly.

For the bile-acids, Pettenkofer's test is used. To two drachms of urine a small piece of lump sugar and strong sulphuric acid are added, the latter slowly, and drop by drop, in order that the temperature of the fluid may be kept as low as possible. At the line of contact of the two liquids a deep purple hue develops itself if the acids are present; if there are none, the colour is reddish or brownish red.

Tyrosine and leucine are demonstrable by microscopic examination of the crystals produced in urine which has been carefully evaporated to the consistence of syrup.

In cases of Jaundice with obstruction, but less so in those without it, there is a deficiency or total absence of bile in the alimentary canal, causing a change both in the colour and the composition of the fæces. The bowels are generally constipated or sluggish. The stools are whitish or white, or clay-coloured or grey, if the food is of the ordinary character; but, of course, many kinds of food or medicines, as iron, mercury, &c., give some colour to the fæces, and this should be remembered when the evacuations are examined.

[1] Harley, op. cit., pp. 58–89.   [2] Op. cit., p. 426.

When the obstruction is partial there may be a tinge of yellow in the stools; and, of course, when the obstacle has given way there may be bile in the fæces long before the skin becomes of natural hue. As already mentioned, the absence of bile in the stools is less marked in the non-obstructive forms: in pyæmia, for instance, they are generally yellow or of light yellow colour.

The fæces in Jaundice when free from bile are generally acid, have a disagreeable fœtid smell, and may contain fatty acids which require bile for their emulsion; and when there is conjoined absence of the pancreatic secretion the neutral fatty matters may also be found unchanged.

There is little or no pyrexia in most cases of Jaundice, but it may occur in the catarrhal or congestive forms. The pulse is generally slow, sometimes descending as low as, or lower than forty or fifty beats. In some fevers the Jaundice is preceded by a considerable falling of the pulse, which is probably due to the depressing effect of bile upon the heart. In some of the pyrexial cases there is fulness and enlargement of the liver.

These preliminary symptoms are sometimes very slight, and diminish in the course of the disease, but do not necessarily subside with the appearance of the Jaundice. Itching of the surface is a frequent derangement; according to Frerichs it occurs in about one-fifth of the cases. It is sometimes very irritating and difficult to relieve; it generally, but by no means always, ceases when the Jaundice is fully developed.

Among the rarer symptoms of Jaundice are yellow tears, sweat and milk, and, still more rare, yellow vision. Elliotson[1] relates a case in which there was yellow vision in one eye, but not in the other. The eye with yellow vision was affected with albugo, through which and across the line of vision ran two large vessels. Diarrhœa instead of constipation is sometimes met with.

The time which elapses between the establishment of the exciting cause and the appearance of yellowness of the surface varies. In some instances it comes on rapidly; cases are related of its manifestation immediately on the occurrence of the mental emotion which gave rise to it. It has been suggested that in these rapid cases less pigment passes off through the kidneys than usual, and it seems that in those in which it passes off in large quantities, the Jaundice may be delayed. We may suppose from Frerichs' data that in the obstructive cases the Jaundice is visible on the surface in about three days from the occurrence of the stoppage, though the pigment is seen earlier in the urine. It occurred within twenty-four hours in a case known to the author, in which it succeeded severe and prolonged sea-sickness in a voyage of nine or ten hours' duration.

In the early stages, the urine is sometimes lessened owing congestion of the kidneys, and there is weight and uneasiness about

[1] Principles and Practice of Medicine, p. 102.

the loins, but these symptoms generally soon subside ; occasionally albumen appears in the urine.

In favourable cases without permanent obstruction, after a few days' duration the disease yields, free discharge of bile returns, and the stools assume their natural colour; the digestion becomes good and the patient convalescent, though he often remains languid and thin for weeks, the yellowness slowly disappearing. Jaundice, however, may be a much more serious disease than this. The cholæmia, or associated blood derangements, may become greater, and the effect upon the system dangerous or fatal ; causing convulsions, sopor-delirium, or coma, which may end in death ; or there may be other alterations of the blood showing themselves in petechiæ, purpura, ecchymoses, extravasation of blood, or hæmorrhages from mucous or other surfaces ; and, owing to impaired digestion, imperfect nutrition, great emaciation, and death by asthenia, typhoid symptoms, or collapse.

While the urine is copious and the kidneys healthy, the biliary matter passes largely out of the system—sufficiently so, indeed, in slight cases to prevent the surface from becoming yellow. If the kidneys fail to act, the bile and renal excretions, and probably various kinds of effete matter, accumulate in the blood, and the before-mentioned most dangerous consequences ensue.

Signs of irritation of the mucous membrane of the intestines, with evacuations of dysenteric character, and hæmorrhage from the stomach and intestines, sometimes occur with great portal congestion both in the prolonged and in the more acute cases.

The causes of death in Jaundice may be summed up as

1. Those affecting the nervous system, delirium, convulsions, coma.

2. Typhoid symptoms, asthenia, &c., exhaustion from impaired nutrition.

3. Hæmorrhages from the intestinal and other surfaces.

There is great difference in the course of Jaundice. In some cases, as in the obstructive forms, it may last for years, the patient preserving a considerable degree of strength and vigour throughout. In others, generally in the non-mechanical forms, it is fatal within a few days or hours. The cause of the differences of symptoms of these cases is of great interest, and is by no means understood. The bad symptoms are not due to the mere presence of colouring matter, as this may circulate freely in the system for years without causing much mischief. It is probable that there is some end to the toleration of this substance, but it is difficult to say where it terminates.

Dr. Harley attributes the bad symptoms to the accumulation of bile-acids in the blood.[1] He found that six grains of glycocholate of soda injected into the femoral vein of a small dog killed it. The symptoms are not given. The experiments of Professor T. H. Albers of Bonn on frogs, showed that in these animals injections

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[1] Harley, op. cit., p. 39.

of small quantities of glycocholic acid under the skin produced cramps, severe spasms, and death.[1] Other experimenters have found that animals operated on by injections into the veins died suddenly without assignable cause.

The results of these experiments, however, have not been uniform. Frerichs,[2] for instance, found that in his experiments the animals suffered very little from the injections of the bile-acid. Nevertheless, with all these discrepancies in the results of experiments, unless there is some great error, it does seem that the bile-acids may be injected into the blood-vessels without serious disturbance.

The quantity of the acid tolerated seems equally uncertain. If the bile-acids are the cause of the violent symptoms, it would be difficult for the advocates of Jaundice from suppression to account for the severe and often fatal consequences which so often occur in this class of cases.

Dr. Budd[3] has suggested that the retention and decomposition of excrementitious matters from which probably the acids are formed, or decomposition of the retained principles of bile, or the development of some new animal poison in the liver, may give rise to the dangerous symptoms; but of this there is as yet no proof.

Dr. Murchison says that the severe cerebral symptoms which occur in those cases of the so-called suppression of bile are probably due to imperfect metamorphosis of the materials from which the urinary solids are derived, and which seem to require the presence of bile in the blood to complete them. Urea is not formed in sufficient quantity, and substances, such as leucine and tyrosine, of intermediate composition between it and the proteine compounds, accumulate in the blood and appear in the urine.[4]

The emaciation and anæmia attendant upon prolonged Jaundice probably depend upon the absence of bile in the intestines, and the consequent interference with the emulsion of food and absorption of nutriment by the lacteals.

From defective absorption and nutrition probably depend the change of the blood in the chronic forms, but this would hardly account for its rapid impoverishment and solution in the acute cases in which these blood-changes appear so speedily.

POST-MORTEM APPEARANCES.—The morbid changes connected with Jaundice, irrespective of the cause of the disease, are to be found in the liver itself, and in distant fluids, tissues, and organs. In this section the condition common only to all forms of Jaundice will be mentioned. The state of the liver itself varies with the different kinds of Jaundice, and will be noticed under their respective heads.

The serum and clot of the blood are yellow from bile-pigment

[1] Virchow's Archiv, 1862, p. 582. [2] Frerichs, op. cit., vol. i. pp. 394, 39
[3] Budd, second edition, pp. 263, 264. [4] Murchison, Clinical Lectures, p. 21

the fibrine in the advanced stages of the disease soft and badly coagulated; the altered condition of the blood is evident in the petechiæ, ecchymoses, and extravasations of blood, found in various parts of the surface and organs apart from the seat of congestions. The bile-acids are not traceable in the blood, having been probably rapidly decomposed therein. Nevertheless, if, as asserted, they are to he found in the urine, in obstructive cases, they ought not to be absent from the blood in such cases during life. Frerichs states that he and Dr. Valentin had frequently examined blood obtained during life by venesection, and also that from the dead body, without finding the acids. The white globules are generally in excess, the red globules are commonly large, with a tendency to adhere together and looking as if acted upon by some chemical agent. Nearly all the tissues are yellow. The skin is most affected, the pigment being deposited in the deeper layer of the epidermis, the sweat-glands being deeply stained. The colour is well marked throughout in the adipose, serous, areolar tissue of blood-vessels, lymphatics, the substance of the bones and teeth ; the brain and nerves are less frequently dyed, and the mucous membrane very seldom.

The kidneys are affected not only by being coloured but by morbid changes due to irritation of, and deposits in, the parenchyma. In the earlier stages they are often enlarged and congested, and the secreting cells loaded with bile. According to Frerichs, in chronic cases the kidneys become of an olive green colour; the uriniferous tubes coiled up and of dark colour, the pyramidal tubuli brown or sap green, or black from deposit. When the deposit is most intense the uriniferous tubes may be observed to be distended with a coal-black brittle mass which may or may not be soluble in caustic potash, or there may be cylindrical masses of amorphous material, brown in the centre but becoming gradually pale towards the periphery. Caustic potash causes them to swell up like coagulated fibrine which has been retained a long time in the uriniferous tubes; the pigment may be deposited throughout the substance of the kidneys.[1] Dr. Harley[2] describes the surface of the kidney after removal of the capsule as having the appearance of being sprinkled with ink when the renal capillaries are choked up, the black specks varying in size from the minutest visible form to a pin's head. In the drawing of a case given by him the kidney is represented as very much enlarged and studded with small abscesses.

In Jaundice of pregnant females the fœtus becomes jaundiced also if sufficient time be allowed. Frerichs states that he has not noticed it in cases in which the mother's disease has lasted from five to fourteen days.

VARIETIES OF JAUNDICE: JAUNDICE WITHOUT OBSTRUCTION.

JAUNDICE WITH CONGESTION OF THE LIVER.—This occurs in the active and passive forms.

[1] Frerichs, vol. i. p. 102, and Atlas, part i., plate i.   [2] Harley, op. cit., p. 57.

Active Congestion.—Jaundice of this class is met with in warm climates and in the warm weather of temperate climates, but its occurrence is not restricted to hot weather. The symptoms are more or less uneasiness or sense of fulness or weight about the right hypochondrium. The liver is always more or less, sometimes considerably, enlarged so that its edge can be felt below the margin of the ribs, but its size is not always sufficiently increased for this, though its increase is recognisable by percussion. There may be uneasiness or pain, sometimes pretty severe, increased by pressure or by lying on the left, and sometimes even by decubitus on the right side, and pain in the right shoulder or under the right shoulder-blade. A bitter taste in the mouth is often perceived, and nausea, furred tongue, and the other signs preceding Jaundice. The stools may be pale or white, but sometimes contain bile and are sometimes loose. The Jaundice begins in two or three days after the onset of the symptoms. There is in most cases some pyrexia and quick pulse. This disease is generally not severe, but it occasionally runs a dangerous or even fatal course. The patient often emaciates during its progress, and his convalescence is slow. This form of Jaundice is frequently complicated with congestion of the mucous membrane of the biliary passages. In some cases it is very difficult to distinguish it from the Jaundice which comes on in fevers or from the catarrhal forms of icterus. The congestion sometimes affects part of the liver only.

Passive Congestion.—This is the consequence of disease of the heart or lungs. It is occasionally met with in acute thoracic diseases, causing retardation of the passage of blood through the lungs or heart, as in extensive pneumonia or pericarditis with effusion. It most commonly occurs, however, in the advanced stages of chronic heart-diseases. The Jaundice is often well marked and persistent; the stools are not always pale. This variety of icterus belongs to heart or lung disease, and need not be further treated of here.

Jaundice from congestion of the liver is included in the section of non-obstructive disease, in compliance with the usual opinion which places it in the class of suppression; but it seems probable that many of these cases both of acute and chronic character ought really to be placed among the obstructive] forms. With enlargement of the liver there is more or less pressure on, and narrowing of, the canals, and probably some alteration of their direction. If in this condition secretion of bile goes on, and in some stages of congestion it is freely secreted, it will be more or less retained sufficiently long in the passages for considerable absorption to take place. This might not be apparent after death, and on examination the bile-passages might seem to be of natural calibre and pervious. It is true that Jaundice does not necessarily occur in all forms of enlarged liver, as in the waxy or fatty form, but in these secretion is probably much less active than in some stages of the merely congested liver.

JAUNDICE FROM MENTAL EMOTION.—There are numerous cases on record in which this appears to have occurred. The mental emotions which have been known to bring on Jaundice are grief or anxiety, anger or fright. It has been reported in certain cases to have appeared very suddenly, immediately upon the occurrence of the mental emotion. The older writers supposed it to be due to spasm of the gall-ducts or common duct. It is, however, hardly possible to conceive a spasm of the common duct which would be persistent enough to arrest the bile sufficiently long for absorption. This Jaundice would be attributed to suppression of secretion from deranged nervous influence by some writers, and by Frerichs and others to increased formation and absorption of bile, or to diminished metamorphosis from the same cause. Dr. Bence Jones shows how this form of Jaundice might arise owing to increased formation of bile through nervous disturbance, especially if at the same time there was interference with the condition of the blood. Referring to the experiments of Claude Bernard on the secretion of the salivary gland, he says: "It is very probable that the sympathetic nerve in the liver if tetanised would stop the circulation by contracting the small blood-vessels, and this would diminish the secretion of bile, whilst the branches of the pneumogastric which enter the liver, when stimulated, would relax the small blood-vessels and thus cause a more rapid circulation through the liver, from which an excessive formation of dilute bile would result."[1] Dr. Murchison thinks that this Jaundice is due to diminished metamorphosis in the blood. The disorder has appeared more frequently when the cause has operated directly after a meal, and, preceded by a sense of weight or uneasiness at the epigastrium and a feeling of sinking and difficulty of breathing. Bamberger attributes the symptoms to a nervous disturbance of the stomach and duodenum which causes a catarrhal state of the mucous membrane of the duodenum.[2] It seems impossible that the rapid icterus could be produced in this way. The yellowness generally subsides after three or four days, and the disease is usually a light one; but it has been known to terminate fatally, with convulsion or other lesion of the nervous system. It appears that in some fatal cases the liver has undergone changes similar to those met with in the graver forms of icterus to be mentioned presently.

MALIGNANT JAUNDICE.—There is a group of cases of very fatal nature characterised by the rapid progress of the worst symptoms of the disease. They are described by Dr. Budd as cases of "fatal Jaundice," by Lebert[3] as "Icterus typhoides," and are mentioned also by Graves.[4] Many of these cases were connected with some stages of that condition of the liver known as acute yellow atrophy, and must be left for consideration in that article, but as they are not

[1] St. George's Hospital Reports, 1866, page 193.
[2] Virchow's Handbuch der speciellen Path. und Therapie, vol. vi. part i., 2nd half.
[3] Virchow's Archiv, vols. vii. and viii., 1855.
[4] Clinical Lectures by Neligan, page 633.

necessarily connected with that affection, it will be fitting to mention the characteristics of the group here.

These cases may begin without severe symptoms, and without any apparent reason pass into the most dangerous conditions. In others the dangerous symptoms show themselves from the first.

There may be pain and uneasiness in the hypochondrium or epigastrium after or with slight feverishness, and then, after a short interval, rapid onset and progress of Jaundice with coma and other symptoms of disturbance of the nervous system, hæmorrhages into the intestinal canal, serous cavities, or other tissues, petechiæ, and · extravasations of blood into the cellular tissues. The blood-changes come on very rapidly, and are characteristic. Head-symptoms are very frequent, and generally set in early. Coma does not always occur; a certain number of cases die under typhoid symptoms or exhaustion. In the beginning of the disease the pulse may be slightly accelerated, but as the illness advances it gains in frequency and loses strength, and in the last stages is subject to great fluctuations within the twenty-four hours. Nausea and vomiting are frequent, and in some instances the vomited matters are dark and grumous, resembling what is known as black vomit. The bowels may be constipated, but diarrhœa is not very uncommon, the evacuations pale or clay-coloured, but they are not always devoid of bile. Taking the three classes of symptoms which attend Jaundice, it seems that they are all intensified and rapid in their advent and downward progress; as might be expected, a very large proportion of these cases die, so that the disease deserves the name of fatal or malignant Jaundice. Its duration is generally short ; few of the cases which end fatally survive the third, but probably the majority of them die within the first, week. Some of these cases are, doubtless, attended with acute atrophy, as the liver is found diminished in size, but many of them of a severe character, and even of some duration, are unattended with change of form or size of the liver. In the fatal cases the tissue of the liver is soft and easily torn, all the gall-ducts pervious, but without biliary secretion or containing thin green bile only ; the cells in the greater part of the liver, and frequently throughout, more or less altered, more or less indistinct or broken down, sometimes quite untraceable, and such cells as can be distinguished not dyed with bile. This severe affection most frequently attacks people below the middle age, and is occasionally met with in the earlier stages of pregnancy. It has been confounded with yellow, bilious, and typhoid fevers. Excellent illustrations of this form of Jaundice may be met with in the works already mentioned, and to them the reader is referred for further information.

The description of Jaundice occurring in the forms of blood-poisoning met with in the course of fevers, pyæmia, poisoning by phosphorus, &c., finds place under these diseases ; to describe them in detail would occupy too much space here. In most of them there is some disordered circulation in the liver; in some, as in Marsh fevers, there may be congestion and enlargement ; in typhus, typhoid and relapsing, and other fevers

of Europe, the probability is that there is no great change of size. In poisoning by phosphorus there is fatty degeneration.

Dr. Morehead met with 28 cases of Jaundice in 114 cases of remittent fever in natives of India. In some of these there was enlargement of the liver and biliary congestion with tenderness below the margins of the seventh, eighth, and ninth ribs. In six of the cases there were traces of inflammation of the mucous membrane of the duodenum or stomach. He concludes that inflammation of the duodenum has an important relation to Jaundice in remittent fevers, but he has not found evidence of congestion or inflammation of the biliary passages.[1] The late Mr. Twining attributed Jaundice in many of these cases to the pressure of certain neighbouring enlarged glands upon the common duct.[2] Dr. Morehead, however, has not seen confirmation of this view in his cases, because, though he found these lymphatic glands enlarged in six out of ten cases, he saw no reason to suppose that in any except one had they exerted any pressure upon the duct.

Virchow thinks that the Jaundice in pyæmia, typhus, &c., depends upon catarrhal states of the biliary canal, and therefore belongs to catarrhal icterus. He believes that the portion of the common duct which runs between the coats of the duodenum, and which he calls the intestinal portion of it, plays the chief part in producing the Jaundice. He says that it is often closed during life by œdema and turgescence which disappears after death, or it is obstructed by a plug of colourless mucus which may be squeezed out by gentle pressure on the duct, but not by pressure on the gall-bladder; the passages behind the plug are widened and stained with bile, the intestinal portion being narrowed and pale. It is quite possible that many cases have occurred, in which morbid changes in the bile-passages were evanescent or not recognised owing to hasty observation, which may really have been instances of catarrhal icterus coming on in the course of the primary disease, and should be considered as belonging to the obstructive, instead of to the non-obstructive class.

A full consideration of the morbid changes in cases in the class of Jaundice without obstruction shows that there may not always be such an absence of obstruction as is generally supposed. In some of the cases of blood-poisoning it is likely enough that no impediment existed at any time, but the number will be considerably reduced if the congestion cases be looked upon as mere instances of arrest of bile in the passages, or, at any rate, of sufficient delay therein for absorption to occur, and if the catarrhal states be proved to be more frequent and effective than is now considered to be the case. If the Jaundice depended upon deficient metamorphosis only, it ought, one would think, to be more frequent in cases of malignant fevers than it seems to be. It is evident that in many cases of fever the catarrhal state has an important effect in producing the result. In many cases of blood-poisoning the secretion of bile, or, at any rate, of its coloured part, goes on. How far this may be due to the action of a part of the

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[1] Researches on Diseases of India, 2nd edit. p. 97.   [2] Twining, Diseases of India.

liver only has not been ascertained.   This continued secretion appears pretty certain, because the gall-bladder is frequently found full of bile after death, and in some cases there is biliary congestion, as, for instance, in many of Dr. Morehead's cases.   In some instances, however, there does appear to have been a deficiency of secretion.   The condition of the liver itself in these cases without obstruction varies.   It may be enlarged, much diminished, or of about normal size, its texture soft and friable and easily broken down, and the colour pale or mottled, the cell-structure broken or disintegrating, not bile-stained or saturated, as in obstructive Jaundice.   Virchow considers that the absence of pigment in the cells would be indicative of want of obstruction.   It would be found in the uninjured cells in cases in which obstruction had come on from catarrh.   Leucine and tyrosine are often found in the substance of the liver.   An acid state of the liver was found in a case related in Dr. Budd's work.[1]   The spleen is often enlarged and softened, and the kidneys generally altered in size and consistence, bile-stained, and often with excess of epithelium in the tubules.

JAUNDICE WITH OBSTRUCTION.

This may be acute or chronic, temporary or permanent, partial or complete.   The most characteristic signs are met with in the complete and permanent forms.

The acute forms are well illustrated by the cases in which the passage is obstructed by swelling or turgescence of the mucous membrane of the bile-ducts, or of the duodenum along the tract on or about the mouth of the common duct; also where a gall-stone slips forward into the common duct, bars the passage for a time, and either passes onwards into the intestines or slips back into its bed, in either case freeing the channel.   In these cases Jaundice is both acute and temporary.   It may be intermittent when a calculus at intervals slips backwards and forwards, or when a series of calculi pass onwards at sufficient intervals of time.   The complete form is illustrated when a gall-stone becomes permanently impacted in the common duct, or when some tumour or morbid tissue constricts the duct entirely, preventing the passage of bile.   The partial form of retention may exist when a gall-stone only partially blocks up the common duct, allowing part of the bile to pass onwards; or when it completely stops up one of the large branches of the duct, imprisoning the secretion of a section of the liver only.

PERMANENT OBSTRUCTIVE JAUNDICE.—When this has become fully established the liver undergoes extensive changes; at first enlarging and afterwards diminishing in size.   The stage of enlargement is due to the accumulation of bile in the ducts and gall-bladder, and in congestion of the tissue.   The liver enlarges in all its dimensions

[1] Op. cit., p. 258.

edge with sharp outline may be readily felt below the ribs, and its depth of dulness, in the sternal, mammary, and axillary lines, evidently increases. In extreme cases the measurement in the mammary and axillary regions may reach respectively two, three, or more inches above the normal standard, and the edge may be felt below the level of the umbilicus. In some cases the gall-bladder attains a great size, and may be easily recognised by the eye and by touch, as a round tumour projecting under the margin of the liver. Frerichs has not found it to contain more than eight to sixteen ounces of fluid ; but larger quantities have been met with. Under the great distension the gall-ducts become dilated and thin, the diameter of the main duct being sometimes enormously increased, and the whole of the ducts throughout the liver may be distended.

After remaining stationary or variable for perhaps five or six months, the liver begins to diminish, the secreting tissue becomes impaired and atrophic. By degrees bile ceases to be secreted, while at the same time the contents of the ducts are absorbed and partly replaced by thin secretion containing little bile. The diminution of the liver may be traced by percussion. In the later stages the organ is reduced below the natural size, and in extreme cases, owing to atrophy of the secreting tissue, may be converted into little more than a cyst.

Symptoms.—This form of Jaundice may set in with marked symptoms, as in the case of gall-stones or obstruction by entozoa, or it may come on slowly without marked signs, as in compression by tumours. It may be attended with the signs of cholæmia already stated. The yellow colour is usually deep—deeper and fuller than in the non-obstructive forms, gradually increasing for some time, but afterwards lessening. The appetite and digestion are often very good at first. The bowels are generally constipated, and the stools white and free from bile. In the later stages there may be portal congestion, dysenteric stools, and hæmorrhages from the intestinal canal, independent of the general hæmorrhagic tendency from impaired nutrition. The Jaundice sets in quickly after the complete establishment of the obstruction, within three or four days. Under favourable circumstances the disease may run a very chronic course, in some cases lasting for years. From imperfect nutrition, the patient may emaciate and waste, owing to the complete absence of bile in the intestinal canal; the blood may become impoverished by degrees ; and, as the secreting tissue of the liver wastes, acholia may ensue.

The symptoms of the associated cause will, of course, vary greatly with the nature of the obstruction. This will be referred to in the paragraphs on diagnosis. Though in occlusion of the ducts in non-malignant disease the downward progress of the case may generally be slow, yet it may become rapid from accidental suppression of urine, or from symptoms affecting the nervous system, hæmorrhage into the bowels, or peritonitis from rupture of the gall-bladder or ducts. The prognosis is also affected by the nature of the associated disease.

Morbid Anatomy.—The changes found in the liver after death depend of course upon the stage of the disease at the time. In the earlier stages, say where the obstruction has lasted a few weeks, we find it much enlarged in all directions, of olive green or brownish green colour, or mottled from bile-staining; the ducts distended with secretion; the hepatic cells grouped round the intra-lobular veins, tinged or clotted with bile-pigment; the nucleus sometimes dyed yellow; the peripheral cells of the lobules pale or scarcely tinged; sections of the liver have a marbled appearance, from the different degree of staining of the central and peripheral hepatic cells.[1]

If the obstruction has lasted longer, say for six or more months, the liver is deeper and more uniformly coloured. The peripheral cells have also become coloured; the finest capillary ducts are filled with bile. In these are deposited minute microscopic bodies, mostly rod-shaped or branched, which lie in the minute network of the capillary ducts and sometimes form casts of the capillary network. With still longer stasis in the capillary bile-ducts the bile crystallizes in irregular, ruby red, shining bodies, which have not the form of crystals of hæmatoidin.[2] The interlobular ducts contain inspissated bile mixed with epithelial cells from their walls, forming small concretions, and the ducts are often lined with a compact tubular dark brown mould or cast,[3] while the duct-walls are thickened. The enlarged ducts can be followed throughout the substance and even on the surface of the liver, and are often varicose or irregularly dilated. The degree of dilatation of the ducts is sometimes enormous; thus Frerichs mentions a case in which the diameter of the common duct reached one and a half inches in diameter, the cystic duct eleven Paris lines, the hepatic duct twenty-two, and one duct in the right lobe seventeen.[4]

In old cases, and when atrophy of the secreting tissue has taken place, the gall-ducts and passages contain a clear slimy mucous secretion, which has replaced the absorbed bile, and, indeed, in some instances the fluid has quite lost all the chemical reaction of bile. When destruction of the secreting tissue has far advanced the liver has a shrivelled or collapsed appearance, and the weight is considerably reduced.

The condition of the hepatic cells in permanent obstruction differs from that in the non-mechanical Jaundice. If examined sufficiently early after death, say within twelve or eighteen hours, they are not found broken up, but they may be granular, and the nucleus obscured. In advanced cases they shrivel up so that the largest cells found are smaller than the smallest normal ones. They have generally lost one-half or one-third of their natural measurement.[5] Decomposition sets in with unusual rapidity after death in Jaundice, and hence unless the liver is examined immediately after death, the cells will be found to be either altered or broken up. This shrivelling-up of the

[1] Frerichs, op. cit., vol. i. p. 131.   [2] Wyss, Virchow's Archiv, April 1866, p. 55.
[3] Frerichs, vol. i. p. 116.   Op. cit., vol. i. p. 139.
[5] Wyss, Virchow's Archiv, April 1866, p. 359.

cells is not always found. Dr. Budd[1] gives an instance of obstructive Jaundice from closure of the common duct, in which the cells were examined by him in the early spring, twenty-two hours after death, and found to be broken up; the liver-tissue contained numerous oil globules. In these cases probably disintegration and suppression had come on in the course of the obstructive disease.

JAUNDICE WITH OBSTRUCTION FROM CATARRHAL STATES OF THE DUO-DENUM OR BILIARY PASSAGES.—These affections are sometimes met with singly, but they are often combined in the same case, the congestion commencing in the duodenum and spreading to the bile-passages. When commencing in the duodenum, the Jaundice is preceded by uneasiness at the epigastrium increased by pressure, dyspepsia, nausea, flatulence, bitter taste in the mouth, furred tongue, loss of appetite, and general debility, and often relaxed bowels, with some feverishness. These symptoms exist for a few days, sometimes for two or three weeks before the obstruction is established and the Jaundice appears. The congestion and swelling of the mucous membrane of the duodenum shuts up the mouth of the common duct. When occlusion is complete, the stools are clay-coloured or white. This disease often spreads along the common and other ducts. When the congestion begins in the passages the symptoms are more obscure. There is less disturbance of the gastric functions, but there is uneasiness in the right hypochondrium, some pyrexia, and sometimes enlargement of the liver from biliary and vascular congestion and feverishness. When the inflammation involves both sets of tissues, the symptoms are a combination of those of both affections.

The congestion and swelling may last a few days, or even two or three weeks, and then, with subsidence of the turgescence, the passage is set free and the bile resumes its course. Frerichs saw a case which lasted three or four months.[2]

The catarrhal is perhaps the most common form of simple Jaundice, and is often associated with hepatic congestion. It is frequently met with in young people, and is generally a curable affection. A fatal case of the inflammation of the common duct is related by Andral.[3] In this the inflammation produced complete obstruction, distension and rupture of gall-bladder, and death at the end of eleven days. In catarrhal icterus the colour of the surface is generally deep.

JAUNDICE FROM THE ENTRANCE OF ENTOZOA OR FOREIGN BODIES INTO THE COMMON DUCT.—This is very rare. Dr. Morehead mentions two cases. It is not easy to diagnose this condition. Inflammation is produced in the passages by the entrance of these bodies, and the symptoms are pain, sometimes severe, in the hypochondrium or epigastrium, with more or less pyrexia, and less disturbance of the functions of the stomach or duodenum than in catarrhal icterus. Hydatid cysts have

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[1] Op. cit., p. 211.   [2] Op. cit., vol. ii. p. 443.   [3] Budd, 2nd edit. p. 184.

been known to open into the gall-bladder or ducts, and to produce obstruction by hydatids being lodged in the common duct.

EPIDEMIC JAUNDICE.—There is no special form of icterus with this character. It is not a very unusual circumstance to hear of several cases of Jaundice occurring in succession in a family, but in other instances the disease has appeared in a decidedly epidemic form in a district. Several severe epidemics, and some of them of a very fatal nature, are on record. In some of them, women in the early months of pregnancy suffered in a larger proportion than the rest of the community.

Epidemic icterus is most frequently prevalent in warm climates, or in warm summers in temperate climates, but is not confined to these; a widely-spread epidemic occurred at Rotherham in the winter of 1862-63.

A few of the epidemics may be noted. Dr. Watson mentions the prevalence of several cases of icterus in 1849. Dr. Morehead notices a sort of epidemic in the 1st Bombay Fusiliers in 1844. One occurred in the Island of Martinique in 1858. Numerous epidemics are mentioned by Frerichs, Lebert, and Copland.

As above mentioned, no particular form of Jaundice can be called epidemic. Generally, however, it has some connexion with blood-diseases, or with gastro-duodenal catarrh, or congestion of the liver. Probably the two latter conditions are the most frequent causes, so that epidemic icterus is frequently connected with temporary obstructive disease, the gastro-duodenal catarrh being part of some general disturbance of the system due to constitutional causes.

JAUNDICE WITH EXCESS OF BILE : POLYCHOLIA.—This is probably not frequent, but occurs sufficiently often to require notice. In this there is excessive secretion of bile, and excessive absorption without mechanical impediment, so that the blood becomes impregnated with bile faster than it can be got rid of by the secreting organs. In this, with the usual symptoms of Jaundice, there is bile in the evacuations, and sometimes bilious diarrhœa. Dr. Murchison thinks that the Jaundice is greater when, with excessive secretion, there is constipation. He attributes the Jaundice to the presence of more bile in the blood than can be transformed by the normal metamorphosis.[1] This form of Jaundice is sometimes, perhaps not unfrequently, associated with congestion of the liver.

JAUNDICE IN NEW-BORN INFANTS.—This may be due to much the same class of causes as those which operate in the adult; they may be catarrhal conditions of the passages or stoppage by inspissated bile or congenital deficiencies or obliteration of the passages, pyæmia, blood-poisoning, &c., or the result of a cold or vitiated atmosphere. T

[1] Op. cit., p. 403.

symptoms are much the same as those of the adult in the obstructive forms, deeply-coloured skin, conjunctiva, and urine, with white evacuations. It may be remembered that the yellowness of the surface, which is so frequent in new-born children, is really not Jaundice, but due to physiological changes occurring in the blood in the skin. In these cases the conjunctiva and urine are not yellow, and the intestinal discharge is natural. In many instances Jaundice in children is a very serious disease, especially when associated with peritonitis, or enteritis, or phlebitis of the umbilical veins. In these cases oozing of blood from the granulations of the surface of the navel often takes place, and is generally fatal. The blood does not coagulate. Congenital deficiency of ducts is fatal, though life may be prolonged for a few months.[1]

DURATION.—From the foregoing it will have been seen that Jaundice is a disease of very different degrees of duration. The simple or common forms of acute Jaundice run a favourable course of ten days to two or three weeks, and are rarely prolonged much beyond. The graver forms of non-obstructive icterus last a few days; the majority of the cases end within a couple of weeks; but some have been protracted to three or four weeks. Cases with yellow atrophy have ended fatally within two days. The duration is longest in cases of permanent obstruction, especially in the non-malignant forms, when it may extend to many months, or even years, without seriously affecting the system. In these cases the balance of comparative health is probably maintained by free secretion through the kidneys and other organs. Drs. Budd, Graves, and Frerichs mention cases of extreme duration. The class depending on malignant disease are, of course, liable to quicker progress, seldom or never extending beyond five or six months; indeed, the duration of Jaundice constantly depends upon, and is influenced by, the associated conditions of the system. The duration may be accidentally shortened by rupture of the gall-bladder and its consequences.

AGE.—There is perhaps no age exempt from Jaundice. The new-born infant may have it if the ducts or passages are imperfect. Young persons are subject to the gastro-duodenal and congestive form; the middle-aged to the obstructive, from gall-stones or cancer. It may appear in any age when it is an intercurrent affection in the course of fever and blood-poisoning.

DIAGNOSIS.—There can be little doubt about the existence of Jaundice when fully formed. Anæmia, chlorosis, and malignant disease may give a sallow or lemon tint of surface, which may be confounded with the lighter forms of icterus. In these, however, the conjunctiva remains white and the urine free from bile-pigment.

It may be well, and especially when epidemic Jaundice is prevalent,

[1] West, Diseases of Children, 4th edit. Lecture xxxv.

in all cases of persistent gastric disturbance and malaise, to examine the conjunctiva and the urine and the right hypochondrium. By so doing, early indication of the approaching condition may be obtained.

The difficulty in cases of Jaundice is to decide upon the etiology or class to which they belong; to decide between Jaundice with and without obstruction. If hereafter proved to be thoroughly reliable, Dr. Harley's test of the presence or absence of bile-acids in the urine might be used. At present, however, the chief and almost only condition to be trusted to is the colour of the stools. If there be bile in them, there cannot be complete occlusion of the common duct. Those who believe in the suppression of bile as a cause of Jaundice would argue that the stools may also be pale from non-secretion of bile. As a rule, however, it may be considered that pale or colourless stools in the early stages of the disease indicate obstruction to the passage of bile into the intestine. Tyrosine and leucine found in the urine denote non-obstructive disease.

The Jaundice which comes on in the course of pyæmia and other blood-poisonings, as in fevers, malarious enlargements, active and passive congestion of the liver, &c., leaves less room for doubt as to its nature if the patient's history and the state of the hepatic and gastric symptoms be duly weighed. If there be obstruction, it is of the catarrhal form. In the more acute forms of non-obstructive Jaundice not following fevers or recognised blood-poisoning, there may be difficulty in deciding upon the exact character of the hepatic lesion. That related to yellow atrophy may be inferred from the rapidity of course and gravity of the symptoms, the early advent of deep Jaundice of tyrosine and leucine in the urine, and the rapid diminution of the size of the liver during life, as shown by percussion—due allowance being made for the tympanitic condition of the intestines which often coexists. Jaundice from active congestion may be recognised by the uneasiness and tenderness in the right hypochondrium and the enlarged state of the liver, recognisable in many cases both by palpation and percussion. Cases of passive congestion are recognised by the local signs and the history of chest-disease. The catarrhal states are diagnosed by deeper colour, the gastric and duodenal disturbances preceding the Jaundice, some degree of pyrexia, and white stools. In later stages some congestion of the liver often comes on, and there is enlargement of the liver. In Jaundice from congestion the enlargement precedes the Jaundice, or is noticed simultaneously; in catarrhal icterus the enlargement of the liver succeeds the Jaundice.

Nervous Jaundice is recognised by its rapidly succeeding the mental disturbance which originates it, and in the absence of local signs.

There may be difficulty in deciding upon the exact local cause of permanent obstruction. The diagnosis is helped by the history and present condition of the patient; in tracing causes, the condition the liver and surrounding parts should be carefully examined by tot auscultation, and percussion.

A severe attack of pain in the hypochondrium and epigastrium accompanying Jaundice, with a history of previous attacks of acute pain in the epigastrium or hypochondrium without fever, and with or without Jaundice, would lead to the inference of obstruction by a gall-stone; there may, however, have been no previous attack of pain or Jaundice. It is possible that the enlarged gall-bladder may be felt as a small round tumour projecting below the margin of the ribs or the margin of the liver itself: an enlarged gall-bladder indicates obstruction.

A history of an attack some time previously of pain in the epigastrium or right hypochondrium, increased by pressure, with feverishness, and with perhaps slight non-persistent Jaundice and subsequent indifferent health, would lead to the inference of obliteration of the common duct by external inflammation, or bands developed external to the common duct, and ending in contracting upon and compressing the canal.

Past symptoms referable to the duodenum, as pain three or four hours after eating without much vomiting of food, would probably indicate the existence of an old cicatrized ulcer of the duodenum near the common duct, which by contracting had shut up the mouth of the duct.

Indications of a cachectic condition of the body may lead to the suspicion of cancerous tumours or growths pressing upon the common duct. The appearance of melanine in the urine might indicate melanotic disease.[1] Careful local examination of the liver and surrounding parts might throw great light upon the cause of obstruction; we should examine carefully the liver itself, and especially its margin. The neighbouring regions should be carefully scrutinized for the presence or absence of abnormal swellings, aneurisms, masses of fæces, pregnancy. In looking for aneurisms the course of the aorta along the spine should be auscultated, and care is required to avoid mistaking the natural pulsations transmitted by an enlarged liver for aneurisms, an error occasionally made. Some assistance in discovering the seat of obstruction is rendered by the consideration of the degree and manner in which the function of neighbouring organs is involved. Thus a growth which closed both the pancreatic duct and the common duct might be in the head of the pancreas itself; in this case neutral fats would not be digested.[2] Tumours pressing both on the portal vein and common duct would cause both Jaundice and ascites without general dropsy. In obstruction acting on the common duct the liver is uniformly enlarged, and its edge smooth; an obstruction barring a branch only would cause local enlargement. A knotty or tuberous state frequently attends cancer. The position of the enlarged gall-bladder may often be ascertained by touch, and be felt readily two or three inches to the right of the median line, projecting beyond the margin of the liver, and moved by the action of respiration. It is a good plan when examining the size of the liver to place the fingers flat, but not

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[1] Harley, p. 68.   [2] Frerichs, vol. i. p. 137.

rigidly, upon the abdominal walls a little below the margins of the. ribs, and to cause the patient to take slowly a deep inspiration. With the descent of the diaphragm the margin of the liver is brought within reach of the fingers, and its position and size often made out most distinctly when other methods fail. The edge glides in an unmistake-able manner under the fingers; the roundness or sharpness and position of the edge are thus readily recognised. Jaundice from the pressure of cancerous glands beyond the reach and touch would be difficult to make out. It might be suspected when, without any preceding history of gall-stones or perihepatitis, and absence of recognisable tumour, there is quick emaciation or a cachectic appearance.

The diagnosis of entozoa in the duct can never attain certainty; sudden coming on of Jaundice, with pains and signs of inflammation about the gall-bladder and ducts with pyrexia, have been noted in some cases which have proved fatal. In cases of hydatid enlargement it may be guessed that an hydatid has opened into the gall-bladder and that a small cyst obstructs the duct, if with pain and feverishness Jaundice suddenly comes on; and the probability is in-creased if a hydatid should pass with the evacuations and the icterus pass off. With the greatest care there must still be cases in which exact diagnosis is impracticable—such, for instance, as obliteration of the duct by slow inflammation unattended with recognised symptoms, small abnormal growths, or a small scirrhous tumour in the duodenum, as in Frerichs' sixth case.[1]

It will be noticed that in the diagnosis some degree of fever, pain, or uneasiness precedes and attends those forms which are attended with congestion and catarrhal states. Severe pain without fever attends obstruction from gall-stones. There may be some fever in the onset of the malignant forms, but their rapidly bad progress soon decides to what class they belong.

For more minute details of diagnosis than can be given here, the works of Professor Frerichs, Dr. Harley, and Dr. Murchison may be consulted with great advantage.

PROGNOSIS.—Most of the cases of simple Jaundice have a favourable termination, but when the Jaundice is well pronounced there must always be some degree of doubt as to the result. The kidneys may at any time become congested, and ceasing to eliminate effete matter become a source of great danger. Free discharge of deep-coloured urine is of good omen. Sometimes dangerous symptoms set in even in cases which begin to all appearance favourably; still, on the whole, cases of catarrhal Jaundice, or those with slight active congestion of the liver, are of good prognosis. Among the dangerous circumstances are all typhoid conditions, fluctuating pulse, and symptoms involving the nervous system, as convulsions, delirium, coma; also great emaciation, undigested food in the stools, ecchymoses, purpura,

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[1] Harley, p. 55.

hæmorrhages from the alimentary canal or dysenteric evacuations, ascites, diminution of urine, and of the excretion of urea, and the presence of albumen, sugar, leucine, and tyrosine in the urine. Dr Graves thought that bilious stools were generally speaking dangerous. This is probably because they are met with in the non-obstructive forms, and therefore belong to severer constitutional disease; but Dr. Graves thought also that they attended scirrhous or serious organic disease. Jaundice in the early months of pregnancy is often dangerous unless it can be traced to simple catarrhal conditions.

The prognosis of the obstructive forms must depend greatly upon the cause of the obstruction, cancerous being more dangerous than non-malignant growths. In all obstructive disease the possibility of ulceration and rupture of the gall-bladder, and its consequences, must not be overlooked. Deep colour of surface and secretion, succeeded by paleness in obstructive disease, indicates deficient secretion of bile, probably owing to impaired cell-tissue, and is unfavourable.

In all forms of Jaundice associated with other diseases, the prognosis must depend upon the prognosis of the concurrent mischief. This must always be attended to in giving an opinion as to probable progress.

TREATMENT.—The forms of Jaundice which come on in the course of fevers and the various kinds of blood-poisoning, if unattended with congestion of the liver or symptoms of catarrh of the mucous membrane of the duodenum, require treatment adapted to the primary disease and to the vital state of the patient. His condition will probably require tonic treatment, with free ventilation and fresh air, and nourishment and stimulants carefully prescribed.

The malignant forms of Jaundice, though not hopeless, do not seem to admit of much treatment, especially after the nervous symptoms have set in. They demand eliminative measures, and those which have recovered have generally done so under the use of this class of medicines. Dr. Budd recommended a mixture with a combination of half to one drachm of sulphate of magnesia, fifteen grains of carbonate of magnesia, and half a drachm of spirits of ammonia, three times a day, " to keep up the action of the bowels, to neutralize any excess of acid in the system, and to keep up the action of the skin." To promote the elimination of urea and uric acid Dr. Murchison recommends warm baths, hot-air baths, diaphoretics, diuretics,. and colchicum. Hæmorrhage may be treated with gallic acid or tannin, &c., or other astringents; the typhoid state by the usual means.

Those who look upon these forms of Jaundice as due to suppression of bile will probably prescribe medicines to stimulate the hepatic secretion, but it must be remembered that in most of these cases secretion of bile is not arrested, though in many cases it seems diminished in common with other secretions, or with the destruction of the hepatic cell-growth and failure of its function. It is obvious that no

[1] Graves, Clinical Medicine, by Neligan, p. 423.

stimulants to secretion will restore the cells to their integrity. The remedies most relied on to promote the secretion of bile are such as ipecacuanha, mercury, podophyllin, benzoic acid, mineral acids, &c. Of late years great doubts have been entertained as to the power of mercury over the hepatic secretion, and it does not appear to increase it in animals. It probably owes its chief use in Jaundice to its purgative action. Dr. Harley recommends benzoic acid. It should be given in the form of pill three times a day; the dose would be about fifteen grains in the day. Even if no good came of it, no great harm would be done by the administration of hepatic stimulants in Jaundice without obstruction.

Jaundice with congestion or with the temporary forms of obstruction due to catarrhal inflammations requires the local application of leeches, warm fomentations, or poultices over the epigastrium or hypochondrium, the free use of saline purgatives. It is usual in hepatic congestion to give blue pill at bed-time and a saline draught in the morning, the effect being not so much to excite biliary secretion and thus relieve portal congestion. The diet in this state should be simple and unstimulating; hot irritating condiments or alcoholic stimuli would be likely to aggravate the hepatic congestion. The treatment of the catarrhal forms is conducted on the same principles. In the milder attacks it is desirable not to do too much. Sparing and simple farinaceous diet, or broths for the first days, diluent iced or effervescent drinks, saline draughts, with alkalies and aperients, perhaps a few leeches, warm fomentations, and rest in bed, are generally sufficient to relieve the complaint. The disease will not yield immediately under any treatment.

Jaundice with excess of bile should be treated with purgatives to remove constipation or to relieve portal congestion. Active congestion of the liver requires leeches, fomentations, &c., to the hepatic region. It sometimes happens that excess of secretion lasts for some time, and then probably depends upon debility. For this, tonics, iron, and quinine should be given. The quinine is especially useful if the complaint has a malarious origin. Good illustrations of the effect of tonic treatment in this state are given by Dr. Handfield Jones.[1]

JAUNDICE WITH CONTINUED OBSTRUCTION.—The object of treatment in this is—1. To remove the cause if possible. 2. To obviate the effects of the absence of bile upon the digestive process and nutrition by remedies and management of diet. 3. To combat such constitutional and local symptoms as may arise, and promote free elimination from the secreting organs. By judicious attention to the second and third objects, life may often be considerably prolonged, though little may be accomplished as regards the first. The cases in which we can hope to do much, or anything, towards the first indications are few. They are such as depend upon fæcal accumulations, hydatids, abscesses, other fluid collections pressing upon the duct. These last will p

[1] Functional Nervous Disorders, pp. 514, 515.

bably be treated surgically on their own account rather than for the secondary disease. Free and repeated purgation by emollient enemata, to which ox-gall may be added, will clear out the bowels and remove the cause of pressure. The passage of gall-stones may be facilitated, and the great suffering caused by them relieved, by opiates given by the mouth or by hypodermic injection. Belladonna is sometimes useful, but less so than opium. Alkaline draughts and warm water soothe the sickness, and perhaps the pain. Warm baths and hot fomentations should be tried, and in severe cases the inhalation of chloroform. For the second object of treatment the bowels should be kept free from offensive matters, but not by irritating purgatives; acidity, flatulence, and fœtid eructations by alkalies, vegetable charcoal, creosote, and the usual remedies. The gastric digestion may be assisted by pepsine given with the meals, muriatic acid and bitters. The food should be selected with care, and such as will be most readily digested by the stomach itself leaves less work for the after stages in the duodenum. A greater quantity of food will be required than in health, because much will be wasted. It should be mostly albuminous and such vegetable matter as does not add to the flatulence. Fatty and oily, and much starchy and pastry matters should be avoided. To supply the place of the bile inspissated ox-gall should be used. The dose should be six to ten grains with each chief meal, given about three hours after it, so as to reach the duodenum about the time that the chyme has passed the pylorus, and thus perform the duty of bile in the right time and place. Dr. Harley thinks that this object is best attained by enclosing the inspissated ox-gall in gelatine capsules, containing five grains each, to enable it to reach the duodenum unacted upon by the stomach.[1] Alcoholic stimulants should be avoided. Claret may be taken if it agrees with the patient; of course, in cases of debility, other wines may be given in moderate quantities. Diuretics and diaphoretics may be given if the kidneys or skin do not act freely. Dr. Murchison recommends the use of the warm bath and the avoidance of sudden chills. The scorbutic, hæmorrhagic, and typhoid states must be met by the usual remedies; cerebral symptoms by purgation, revulsives, and diuretics,—avoiding cantharides as Dr. Murchison judiciously observes;—dysenteric stools by emollient injections or occasional aperients of rhubarb, fomentations, sedatives, and small anodyne enemata; inflammation about the gall-bladder or ducts, by leeches, fomentations, &c.; rupture of the gall-bladder by fomentations and the free use of opium. Careful nursing and cleanliness, and abundance of fresh air, are essentials; bad bed-sores are apt to form in the typhoid stages, especially when the urine passes involuntarily. It is scarcely necessary to notice that medicines which increase the secretion of the liver are injurious in obstructive jaundice.

In order to obviate the destructive effects of long-continued permanent obstruction upon the secreting tissue of the liver, Dr Harley

[1] Op. cit., p. 129.

has advocated puncture of the distended gall-bladder and the establishment of a biliary fistula. The gall-bladder has often been punctured before for biliary distension when it has threatened to burst and permit of extravasation of its contents. It would not be difficult to reach the distended bladder from the surface. Of course, there would be risk of extravasation into the peritoneal cavity, a circumstance very likely to occur after puncture, as the retreating liver would withdraw the gall-bladder from the abdominal walls. Dr. Harley recommends the use of escharotics, to produce adhesions between the gall-bladder and the parietes of the abdomen previous to puncture, in the manner recommended by Dr. Graves for opening hepatic abscess. Some such steps would be necessary before attempting an operation. There would doubtless be danger in this operation, but in permanent non-malignant obstruction such an operation would probably preserve the secreting tissue of the liver, and deserves mature consideration. If a biliary fistula were well established, it might become a question whether, in a case of impacted calculus, dilatation of the opening might not permit or facilitate the escape of a concretion or the extraction of small ones.

Itching of the surface is sometimes very troublesome. It may be relieved by warm baths, friction of the surface, potash or soda internally, and, in severe cases, by opiates.

The yellowness of the skin remains for some time after the cause of Jaundice is removed. Baths with carbonate of soda, benzoic acid, and aperients are supposed to be useful. Probably time and fresh air are the most efficient agents.

The treatment of convalescence must necessarily depend upon many conditions. Residence at the seaside or at Cheltenham, and the use of the Cheltenham waters, are beneficial. It is not necessary, in cases of simple Jaundice, for patients resident in tropical regions to seek change of climate. This, however, should be resorted to if convalescence is prolonged, or if there be serious congestion of the liver or enlargement, the result of malaria.

BILIARY CALCULI.—GALL-STONES.

By Edward Goodeve, M.B.

Description. — Concretions originating in the gall bladder, or biliary ducts, derived partly or entirely from the constituents of the bile. They are met with in all parts of the biliary passages.

These concretions vary in size, shape, colour, specific gravity, structure, chemical composition, and in the number existing in the same individual.

The *number* may be one or many hundreds. There may be a simple concretion, or they may be, and generally are, numerous. The fewer the number, the larger they generally are. They are often found in large numbers in the bile ducts, in very rare instances distributed throughout the liver; in one case they were so numerous as to impede section of the liver with a knife. Though they may exist in any part of the bile containing apparatus, they are far more frequent in the gall bladder than elsewhere, so that this receptacle may be considered as their principal seat.

The *size* and *shape* depend much on the number; when numerous they are generally small. The larger concretions are generally found in the gall bladder; a solitary oval one, moulded to the shape of the bladder, sometimes completely fills it. They have been found as large as a hen's egg. Meckel mentions one of five Paris inches in length by four in circumference.[1] Mr. Blackburn records one of 3¾ inches in length and 1½ in largest diameter, and weighing 1 oz. 6 drachms.[2] Concretions one or two inches long are not rare. When very numerous, the calculi are not larger than a hemp seed or millet seed; when smaller than this they are called biliary sand or gravel; the most common sizes are those compared to peas, or the kernels of hazel nuts. When solitary, calculi are generally roundish or oval; when numerous they are of roundish or squarish outline, with facets, or many sides, from attrition. They are sometimes found filling up the gall bladder, and fitting accurately to each other by their sides. Occasionally, when two or three only exist, they may articulate as it were, by their ends. In rare instances, the con-

[1] Frerichs' Clinical Treatise on Diseases of the Liver. New Sydenham Society's translation, vol. ii. p. 499.
[2] *Lancet*, December 12, 1868.

cretions have a leaflike appearance, with black glistening surfaces, and those found in smaller canals may have a coral, or branched-shape, like casts of their ducts.[1] The surface is generally smooth, especially when they are multiple, but they have sometimes a wrinkled appearance like peppercorns, or are tuberculated or rough, like small blackberries. Good coloured illustrations of Biliary Calculi are given in the second part of Frerichs' Atlas, and in the plates of Dr. Budd's Diseases of the Liver.

Biliary sand or gravel sometimes exists in considerable quantity. Dr. Thudichum found the biliary ducts of a man who died with gall-stones, full of brown sand-like matter. Dr. Handfield Jones relates a case of jaundice and death owing to obstruction caused by them.

The *consistence* may be firm or waxy, they may be cut with a knife without much difficulty, or easily crushed. They have frequently a soapy or greasy feel. They are often brittle, and can be broken down with slight force into sand or gravel. Dr. Thudichum thinks that the material binding the particles together is cholic or choloidic acid, or both;[2] it has also been supposed to be mucus.

The *colour*, externally, may be white or whitish, brownish yellow with greenish tinge, blackish, or mottled; some are very dark or black; the colour depends on the chemical composition. They are generally opaque, but some are pellucid, or slightly translucent.

Structure. — Gall-stones are sometimes homogeneous, but are generally made up of different chemical compounds, arranged in layers, or mixed together; sometimes they have a radiated appearance. They are generally composed of a nucleus, body, and crust, the nucleus being surrounded by the body, to which succeeds a shell or crust, and these parts are often easily traceable, on section, by their colour, in which there are sometimes great contrasts, but these distinct layers are not constant. The nucleus is generally dark, and may be of pulpy, hard, or friable consistence, the body lighter than the nucleus and the external layer lighter still; but this is not always the case, a dark deposit may succeed a light one. The nucleus is more or less round, and generally consists of a small quantity of biliary colouring matter; frequently of the small black shrivelled concretions of deposited bile pigment, which have passed from the smaller bile ducts to the gall bladder, and served as a foundation. Dr. Thudichum has shown that in some cases the central or nuclear part contains casts of the bile ducts, and he has given several coloured illustrations of these bodies.[3] Epithelium sometimes serves as a nucleus. The nucleus is generally small, the bulk of the calculus being made up of the deposits around it. Two or three nuclei are sometimes found; in one instance as many as five existed. In these cases several small calculi or nuclei, being cemented together, have

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[1] Frerichs' Clinical Treatise on Diseases of the Liver. New Sydenham Society's translation, vol. ii. p. 501.
[2] Thudichum on Gall-stones, p. 166.　　　　　　　[3] Ibid. p. 60.

become the origin of a larger concretion. Though the nucleus is usually dark, it is sometimes white, as when it consists of cholesterine. In rare instances, it is some foreign body which has found its way into the ducts, as a fruit stone, a clot of blood, or an entozoon. A case is recorded in which it consisted of a globule of mercury,[1] and in another of a needle.

The body, sometimes called the striated part, is generally radiated, sometimes homogeneous looking, or in layers. It consists of cholesterine, more or less mixed with pigment.

The crust or cortical part consists of successive layers, and is found on calculi which have existed some time; occasionally there is scarcely any body, and the cortical layers surround the nucleus immediately. The crust is more developed in some parts than others; it may consist of cholesterine giving a white colour to the calculus, or of pigment and lime, or of carbonate of lime. In some cases, from unevenness of deposit, the external layer may in one part consist of cholesterine or pigment compound, and in another of carbonate of lime, the latter being probably derived from the walls of the gall bladder. It will be impossible to describe the varieties of appearance presented by the deposition and different parts of these layers.

The *specific gravity* of the concretions differs with the composition. With the exception of those of cholesterine, they are slightly heavier than, and sink in, water; when they swim it is owing to air contained within the interstices of the calculus, after their having been kept for some time in a dry place. The specific gravity varies from about 0·800 to 1·50 or 1·60.

The chief *chemical constituents* of gall-stones are cholesterine, the colouring matters of the bile, biliary acids, fatty acids, carbonic or phosphoric acids, lime, soda, magnesia; with these may be mixed mucus and epithelium. Cholesterine and colouring matter are the most common elements. The difference in kind and proportion of the constituents accounts for the different physical conditions of the stones. Generally, but not without exception, the calculi found in the same subject are of similar composition. Chemical analysis is required to decide upon the minute composition of gall-stones, a full description of which is given in Dr. Thudichum's work. Their chemical nature, however, is roughly indicated by their physical conditions. The white or colourless, pearly, nearly transparent calculi, with crystalline fracture and radiated structure, consist of little else than pure cholesterine. They are generally covered with minute crystals of cholesterine, are soapy to the touch, of little weight, and are often large. Cholesterine, mixed with little or much bile pigment, and a little lime or magnesia, constitutes the majority of calculi. When they contain a large percentage of cholesterine, they are of whitish colour, of light specific gravity, and often have a lamellar arrangement. In others with a larger amount of pigment, the colour is much deeper. This may be partly uniform, or may be distributed in layers which may

[1] Franconneau Dufresne, Précis des Maladies du Foi, &c. Paris, 1856.

be concentric with cholesterine, showing thus alternate layers of white and dark yellow pigment deposit. These are generally of medium size. The small, black, angular, irregular, tuberculated, brittle masses, about the size of and somewhat resembling peppercorns, are composed of bile pigment and lime; the bile pigment is in a modified state, and called by Dr. Thudichum modified cholochrome. The resinous calculi of Dufresne are dark in colour, easily crushed, with fracture like sealing-wax, forming a powder of yellowish-green colour, like powdered aloes; they are very rare, and consist chiefly of cholic acid. Besides these are concretions made up chiefly of stearate and margarate of lime, which are of dirty white colour, and lamellar structure, somewhat like cholesterine calculi. The carbonate of lime species are of crystalline fracture, rough surface, with sharp angles, brown or brownish colour, and heavy. They consist almost entirely of inorganic matters.

Biliary sand or gravel, according to Dufresne and Thudichum, consists of three kinds of ingredients, cholesterine, colouring matter, carbonaceous or black pigment matter. The cholesterine sand is yellowish from bile-staining, facets are often recognizable on them with a lens; pigment sand is in small grains mixed with a little cholesterine; and black sand resembles powdered coal or jet.

Dr. Thudichum has classified gall-stones according to their prevalent chemical composition as follows:—

1. Pellucid or pure Cholesterine Calculi.
2. Mixed Calculi, with prevalence of Cholesterine.
3. Calculi, with prevalence of Cholochrome.
4. Calculi, with prevalence of modified Cholochrome.
5. Gall-stones, with prevalence of bile acids.
6. Gall-stones, with prevalence of fatty acids.
7. Gall-stones, with prevalence of Carbonate of Lime.[1]

CAUSES OF GALL-STONES.—These are obscure, and are to be sought for in local and constitutional conditions. The local are such as lead to chemical changes of the bile within the gall bladder or passage, allowing precipitation of its constituents. Any circumstance which causes an acid state of the bile, or interferes with the solubility of the colouring matter, leads to the formation of a precipitation or nucleus, round which other constituents of the bile may group themselves. The growth is favoured by stagnation of the bile in the gall bladder, and hence the reason, probably, why this receptacle contains more than other parts of the passages. In favour of the view of the precipitation being due to morbid states of the bile, owing to decomposition, it has been found that when bile is allowed to stand for a long time in a bottle, it becomes acid spontaneously, as Drs. Thudichum and Gomp. Besanez have shown, and deposits pigment previously dissolved, probably in the cholate of soda. It is likely that in t

[1] Franconneau Dufresne, Précis des Maladies du Foi, &c. Paris, 1856; p. 107.

healthy bile such a change may occur, lead to a deposit, and originate calculus.

Dr. Thudichum suggests that the decomposition may be due to a putrid ferment, absorbed by the intestine. Mucus from catarrhal states of gall bladder or ducts has been supposed to favour the decomposition. When the commencement is made, if the nucleus is retained long enough in the passages, other matters group themselves around it, and growth ensues, even if the original morbid condition is absent. Carbonate of lime appears to be in many cases, at all events, furnished by the walls of the bladder or ducts. It is doubtful whether inspissation of bile can form a nucleus, or whether it is necessary that there should be any excess of the material forming the calculus in the bile.

The constitutional states which favour the formation of gall-stones appear to be such as relate to life, sex, habits, diathesis, &c.

Age.—They are much more frequent in middle-aged or elderly than young people. The great majority of cases occur above the twenty-fifth year. A few only are found below that age. Wolff mentions a case of a boy of ten years old, one of twenty, and of twenty-two years of age.[1] They have been found in new-born infants. Women are more liable than men—according to some statistics as three to two. Fat people, and those of indolent and sedentary habits, which favour the stagnation of bile in the passages, seem more liable than the active and spare, but these latter are not exempt as most practitioners must have noticed, nor are phthisical people. There is no certain connexion between diet and hereditary tendency and gall-stones nor any known diathesis predisposing to it.

The increase of cholesterine in the blood of old people has been considered as a predisposing cause, but gall-stones form before the period of life in which this change occurs. As cholesterine exists naturally in human bile, a supply of this element is always present, and no special blood-change is needed to furnish it. It cannot be said that any known constitutional state has any decided influence in the production of gall-stones. At present it seems more easy to attribute their origin to changes in the bile from some local cause, their subsequent form being determined by simple deposition, as in the case of a foreign body in the urinary bladder.

CONSEQUENCES AND EFFECTS OF GALL-STONES.—These are various and often dangerous. They may be arranged under the following heads :

1. They may remain stationary in the ducts or gall bladder, without causing much inconvenience.

2. They may pass from the branches of the ducts, or gall bladder, by the common duct into the duodenum, causing in their passage slight or severe disturbance of the system.

3. They may be arrested in their passage, and act as plugs, completely or partly obstructing the channel in which they are stopped ;

[1] Virchow's Archives, 20th vol. pt. i. p. 2. 1861.

sometimes causing obstructive jaundice, or even permanent closure of the duct.

4. They may become encysted, or, at any period of their existence, give rise to degeneration of neighbouring tissues, inflammation, suppuration, softening, ulceration, pyæmia, or even rupture or perforation of the structures in which they lie, leading to extravasation of the contents, or giving rise to fistulous passages, by which the calculus may escape.

5. Arrived within the intestinal canal, they may be voided with the evacuations, and entirely got rid of, or they may be arrested in some part of the canal, causing obstruction or other mischief.

It may be well to consider these consequences *seriatim*.

1. In numerous instances gall-stones have been found in post-mortem examination, without any suspicion having been entertained of their existence. It is evident, therefore, that they may be latent. It is rare, however, that they do not cause some disturbance, though it may not have attracted notice.

2. Though a quiescent calculus in the ducts or gall bladder may cause little inconvenience, it often gives a great deal of pain when it moves onwards. It is especially severe when it passes from the gall bladder into the common duct. Concretions in the smaller branches of the ducts may move onwards without much disturbance, because the channels become wider as they progress. If small they may even pass through the mouth of the common duct, without severe symptoms. Biliary sand or gravel generally passes easily and unnoticed. The pain which attends the passage of calculi through the ducts is known as biliary colic, or as a fit of gall-stones.

3. Simple arrest of a calculus may interfere with the free course of the bile, and its effects vary with the place of detention. Arrested in the cystic duct, it will prevent the bile from passing in or out of the gall-bladder. If the stoppage is permanent, the bile in the receptacle will be absorbed, and replaced by mucous secretion, by which the bladder may be distended, in some instances enormously dilated, and the walls become thin and transparent. When arrested in a small branch of the ducts, the calculus may cut off the passage of bile, which accumulating, may press upon the secreting tissue behind it, and ultimately produce atrophy of the part involved. If the arrest be in a large branch or in the hepatic or common duct, obstructive jaundice will follow. The completeness of the jaundice will depend upon the efficiency of the plug; rounded and smooth concretions more completely fill up the passage, but angular stones often permit some bile to trickle past them, allowing fæces to be more or less coloured. The size of the calculus that can pass through the natural passage is larger than might be supposed. Rokitansky says that one the size of a hen's egg may do so, but there is little doubt that when not larger than an almond or walnut they have escaped through the duodenal orifice the duct. This has sometimes been found, in cases in which stone have passed, large enough to admit easily the forefinger. It is probable

that the very large calculi which have passed by stool have found access to the intestine through fistulous openings.

The passage of one calculus smooths the way for those that may follow. These if not much larger than the pioneer stone, generally pass with much less distress. The disturbance caused by a gall-stone is not always proportioned to its size. Angular concretions cause more pain than smooth ones of similar size. When the gall-stones are numerous, several may pass in a single attack, and as many as a hundred have passed by stool after a fit of biliary colic.[1] Gall-stones in the gall bladder or cystic duct do not cut off the passage of bile, and thus do not cause jaundice,

4. Gall-stones may at any period of their existence cause serious or dangerous symptoms. They may excite inflammation of the part in which they lie. This, in its most mild form, may end in the stone becoming encysted, and thus cut off from contact with bile and growth by deposition. In other instances suppuration or abscess may occur. If the cystic duct is stopped up when suppuration occurs within it, the gall bladder may become greatly enlarged by purulent distension. Repeated attacks of inflammation of the gall bladder may cause it to thicken, and, if there be no contained secretion, to shrink or shrivel up. If there be calculi within it, they may be tightly embraced by the contraction. When inflammation occurs round calculi within the ducts, abscesses may form in the neighbouring parts of the liver, and the calculus may pass from the duct into the substance of the liver, and lie embedded in the abscess. Other results of inflammation are softening or ulceration of the tissues round the stone, which may allow of rupture of the parts and escape of the calculus from its bed into neighbouring parts. In these cases rupture may take place under slight movements, or in coughing and vomiting. If this accident happens to the gall bladder, or hepatic or cystic ducts, extravasation may take place into the abdominal cavity, giving rise to perforative peritonitis. Fortunately, in the more common process by which a calculus escapes from its bed, adhesions are formed between the containing part and some neighbouring viscus or tissue. If this occurs at some part of the stomach or intestines, softening and absorption, slight suppuration or destruction of the intervening tissues takes place gradually, and an opening is made by which the stone, and fluid matters surrounding it, bile, pus, or blood, pass into the tube, and a fistulous opening results. The duodenum is the most frequent seat of this process, but it may take place in the stomach, colon, or small intestine. It has occurred in the portal vein and in the ureters. Sometimes the gall bladder is glued to the abdominal wall, an abscess may form and lead to discharge of the stone through an external opening formed naturally, or by incision. Sometimes the opening or pointing of the abscess is at a considerable distance from the region of the gall bladder. In rare instances the stone has passed into the thorax.

[1] Franconneau Dufresne, *Précis des Maladies du Foi*, &c. p. 334.

In all cases of fistula bile may continue to flow through it, but this will depend upon the connexion with the biliary passages being open or not. Occlusion of the cystic duct will prevent bile passing through the gall bladder, and the same will happen for other parts if the communication with the bile ducts is cut off. When the communication is open, the bile will continue to pass by the new channel for an indefinite time, but if the natural passage is pervious, the cholic fistula may gradually close. In some instances calculi will continue to pass by the fistula for some time.

Pyæmia and secondary abscess in the liver are among the most formidable occurrences that attend gall-stones. The walls of the gall bladder may undergo fatty degeneration in cases of calculus lodged within it, and rupture and extravasation may occur without previous marked symptoms.

5. When the gall-stone has reached the intestine, it is generally evacuated by stool. If it passes into the stomach, it may be vomited. Dr. Murchison doubts whether a large calculus which has entered below the pylorus can pass upwards.[1]

Gall-stones are not always got rid of so easily. It is supposed that they may grow larger within the bowel, if detained within it, as sometimes happens. They have sometimes caused obstruction. Dr. Murchison mentions a case in which the calculus fitted the bowel like a cork, and produced fatal obstruction. Dr. H. Jeaffreson relates one in which perforation of the ileum just above the ileo-cæcal valve was caused after a long interval by a stone passed into the duodenum through an opening in the gall-bladder.[2]

Gall-stones may be entangled in the pouches and angles of the intestines, and have been known to be arrested in the vermiform appendix of the cæcum. The consequences may be inflammation, gangrene, perforation, peritonitis; in short, all the evils consequent on permanent obstruction of the intestine.

SYMPTOMATOLOGY.—It will be sufficient to mention the ordinary or special symptoms of gall-stones; the accidental ones, such as jaundice, inflammation, suppuration, pyæmia, &c., need not be detailed here.

Calculi when in the gall ducts, and even in the gall bladder, unless of unusual size, as already shown, may excite no disturbance, or only uneasy sensations, occasional shivering, with or without heat and sweating, which may be mistaken for ague, and be sometimes manifested periodically in women. Small calculi may pass into the intestine without marked symptoms. Biliary sand or gravel may cause only indefinite symptoms of weight and uneasiness, and often pass away from the liver unheeded. The symptoms produced by the passage of larger calculi are often of the most painful and agonizing kind. Those

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[1] Lectures on Diseases of the Liver, p. 509.
[2] *British Medical Journal*, May 30, 1868.

o the passage of a calculus from the gall bladder through the
duct, are typical of the attack of biliary colic, and will serve
ustration.

these fits, perhaps, after experiencing uneasy sensations for an
or two, or perhaps quite suddenly, the patient is seized with
t pain in the epigastrium or lower part of the chest, coming on
roxysms, abating in intensity, and renewed with increased
ce.   The pain generally comes on two or three hours after a
about the time that the food begins to pass into the duodenum,
e bile to flow from the gall bladder, and may appear to originate
me exertion, or jerk, or coughing, or some such movement.
ting comes on, first emptying the stomach of any contents, and
ards continuing and bringing up very acid clear fluid: it is
imes very distressing.   In milder cases there may be only
ng and nausea.   The attacks frequently begin with shivering,
may be followed by heat and sweating; shivering often lasts
me time.   The most remarkable and characteristic symptom is
in.   In some cases it is most agonizing.

patient describes it as burning, cutting, twisting; he is generally
restless, or rolls in agony on the floor, or presses his hands on the
strium, or seeks all sorts of positions, to obtain momentary ease.
eat of the pain is referred to the hypochondrium, the epigas-
and lower part of the chest, sometimes radiating to the
les, and with sense of constriction over both sides.   Although
is difficulty in defining the seat of pain in many cases, and
ially at the onset of the paroxysms, it is sometimes pretty clearly
ed to the right hypochondrium and epigastrium.   In some
ces convulsive movements of the muscles of the right side of
bdomen have come on; these have extended to the whole of
ght side, and have been known to end in general epileptic con-
ns and delirium.   The sufferer is bathed in cold sweat, the pulse
es feeble or thready, and fatal syncope has occurred.   Generally
lse is not excited, and retardation of the heart has been noticed,
oked upon as diagnostic; but though as a rule slow, it is not
ably so.   There is often much flatulence and generally con-
ion.   The duration of the fits of colic may be a few hours, but
ave been prolonged for two or three days, though this is rare.
may return every day.   Wolff has known them to do so daily
egularity.   The interval between the fits may be days, weeks, or
s.   The fits may extend over a considerable time before the
are got rid of; in Wolff's cases, the longest period was about
n months; the majority of the cases occupied from two to
months.   It may happen that a solitary calculus may be
ed and the disease come to an end, but there is more frequently
ession of attacks, lasting over a considerable period.   As has
nentioned, the fit of pain may come to an end after more or less
ng.   The cessation of the pain may be due to the stone having
back into the gall bladder, to its having passed into the

common duct, or to the exhaustion of expulsive efforts. When the calculus falls back into the gall bladder, the pain may cease for an indefinite time : if merely arrested in the ducts, the pain will probably be renewed shortly. In the latter case the stone moves on towards the duodenal orifice, the narrowness of which opposes a fresh obstacle, bringing back fresh spasm and pain. After a renewal of the struggle the calculus slips into the duodenum with great if not perfect relief, the transition from agony to ease being sometimes very rapid. A sense of soreness, however, sometimes remains for a few days. A succession of calculi passing through the cystic duct or orifice of the duct may keep up a renewal of the paroxysms, but generally the passage of the first is by far the most painful, and as the duct is dilated numerous calculi may subsequently escape with little or no annoyance. Still the pain is very severe and distressing, and may be prolonged with variable intervals for months, being most distressing to the patient and annoying to the practitioner. In the mean time the sufferer's nutrition is pretty well maintained if there is a fair interval between the attacks.

During the paroxysms there is sometimes pain on pressure over the gall bladder and hypochondrium ; in some of these instances the pain is more fancied than real, as the patient often presses the epigastrium himself. After the fit is over, there is often much soreness and tenderness lasting for days.

Biliary sand sometimes causes fits of colic equally as severe as those of calculi. It must not be supposed that all fits of biliary colic are of the severe character just described. The pain is sometimes less marked and the whole fit less exhausting. The degree of pain will vary somewhat with the size and shape of the stone and the susceptibility of the individual. There is also much difference in the degree of the reflex disturbances.

Jaundice is not a necessary consequence of gall-stones even when they pass into the bowel. Impaction in the cystic duct does not cause it, and it will not occur if the calculus is not detained beyond a couple of days in the common duct, but when there is delay beyond this, jaundice and white faeces will ensue. The icterus will be slight, coming on towards the end of the attack and quickly passing off, or becoming permanent if the impaction continues. Jaundice is often considered a necessary part of biliary colic, but observations show that it is absent in a large proportion of cases. Twenty-five of Wolff's forty-five cases passed through the whole train of symptoms without it, the concretions being found in the evacuations.

It is desirable to watch the faeces after these attacks, and satisfactory to know that the stones have passed. The faeces should be well diluted with water, thrown on a sieve with small meshes, by which the calculi will be arrested. Simple dilution and pouring off the fluid a few times will leave a sediment in the vessel in which they may be found. As they occasionally float, the surface should be examined also. It is not enough to watch for a day or two after the

attacks. The presence of gall-stones in the bowels may cause no symptoms. They may be evacuated without any disturbance, or they may cause a little griping and colic, or tenesmus in passing the anus. The symptoms peculiar to irritation or obstruction of the bowels need not be detailed here.

DIAGNOSIS.—Biliary Calculi frequently give rise to no symptoms, so that their existence is unsuspected. This is especially the case in the smaller duct. They may be suspected when there is dull pain in the hepatic region, and occasional fits of shivering, with heat and sweating, resembling ague. In the gall bladder they may also be latent, but are more likely to cause uneasiness. The pain is sometimes dull; sometimes pretty sharp, and referred to the seat of the gall bladder, or edge of the liver. The diagnosis of gall-stones is more difficult in countries in which hepatic disorders are prevalent, but the absence of enlargement of the liver or spleen, or anæmia, or history of inflammation of the capsule, will assist the diagnosis. The dull pain in the right hypochondrium, caused by the distension of the colon, will be removed by purgative medicines or enemata. It is well to remember that fits resembling ague, occurring in persons of fair health, may depend upon gall-stones in the ducts. In cases of pain in the right hypochondrium, the region of the gall bladder should always be explored. Fullness of the gall bladder may sometimes be felt, and a peculiar crackling sensation, indicative of calculi, perceived by the tips of the fingers. A large stone may sometimes be made out pretty accurately. They may, if numerous, sometimes be recognised by the stethescope, if pressure is used at the same time. In fat people the state of the gall bladder is not likely to be ascertained.

Fits of biliary colic are generally not difficult of diagnosis. The sudden accession of pain, aggravated in paroxysms, and attended with shivering fits two or three hours after meals, the constrictive nature of the pain, and its being generally referred to the right side of the abdomen, are very characteristic. Jaundice coming on makes the diagnosis more certain, and the finding of the stone confirms it. In a large number of cases, however, jaundice is absent. The absence of feverishness and quick pulse, the avoidance of movement on the part of the patient, and the catching pain in respiration, distinguish acute inflammation of the capsule, or surface of the liver, from biliary colic. Biliary colic may be mistaken for painful affections of the stomach, intestinal and renal colic, and aneurisms of the abdominal aorta and hepatic arteries.

It may be distinguished from pain of undigested food in the stomach unable to pass the pylorus, by the manifest relief afforded by the vomiting. From simple neuralgia of the stomach by the seat of pain and uneasiness about the gall bladder, and soreness on pressure there during the intervals of the attacks. The painful symptoms of organic disease of the stomach are generally distinctive enough. Intestinal colic is traceable to indigestible food or lead

poisoning, and is felt lower down in the abdomen. Renal colic is accompanied with irritation of the urinary organs, blood disks or decided blood in the urine and drawing up of the testicle.

Aneurisms of the abdominal aorta sometimes give rise to intense paroxysms of pain which may be referred to the right side.[1] Frerichs relates cases of aneurism of the hepatic artery, which were mistaken for gall-stones and which even caused jaundice. Aneurisms may generally be detected by tremor, pulsation, and murmurs. In one of the cases mentioned by Frerichs, however, the aneurism of the hepatic artery was not larger than a pigeon's egg, and hardly diagnosable. Fortunately, aneurisms of the hepatic artery are very rare. In some persons, owing to the wide radiation of pain, and the difficulty they have of localising their sensations, very little assistance in the diagnosis is derived from inquiry as to the seat of the pain, and if jaundice is absent it is difficult to decide upon the existence of biliary colic. It is well to remember that a great number of the painful and spasmodic affections of the upper part of the abdomen, occurring in elderly persons not otherwise unhealthy, depend upon gall-stones.

If the calculus is large with a rough surface, we may hope that it is solitary. If, on the other hand, the calculi are smooth and facetted, they are numerous.

We should infer that a calculus was arrested in the cystic duct if the pain ceased, leaving perhaps uneasiness about the gall bladder, and if after a little time it began to distend without jaundice or enlargement of the liver. If on the other hand, jaundice and enlargement of the gall bladder and liver come on, we should infer impaction in the common duct.

PROGNOSIS.—This must be guarded. A large number of cases recover completely after more or less suffering. Still the accidents which have been described as occasionally attending gall-stones from their beginning until their final expulsion from the body, show that they involve considerable risk. These cases may run on for years and ultimately become dangerous.

TREATMENT.—This should aim at relieving the effects of Biliary Calculi, and preventing their formation.

The violent pain of biliary colic calls urgently for relief. This must be attempted by anodynes, of which opium in some shape or other is amongst the most efficacious. Half a grain of morphia freshly made up into a pill, or in solution, may be given and repeated every two hours for two or three times if the pain does not abate. The effects of opium are most certainly and speedily produced by the hypodermic injection of morphia : one-sixth of a grain may be injected beneath the skin, and repeated every two or three hours till one grain has been administered, but of course the repetition must be guided by

[1] Stokes, Diseases of Heart and Aorta, p. 612.

cumstances and not resorted to until the effects of the previous dose are passing off. If the pain is greatly soothed it will be sufficient. If the opiate treatment does not relieve the pain, chloroform inhalations may be resorted to; the patient may be kept under its influence for some time. It is not necessary to produce deep coma throughout the paroxysm. Chloroform may also be given internally instead of by inhalation. Belladonna is supposed to relax the spasm: it may be given in half-grain doses of the extract, or in ten minims of the tincture every three hours, and withdrawn if its physiological effects begin to show themselves. Belladonna, however, seems more useful in the milder forms of the disease, in which the passage of the calculus is prolonged; given then in half-grain doses, two or three times a day, it may facilitate its passage. Benefit is derived from the use of the warm bath. If there be much depression, however, instead of this, warm fomentations may be applied assiduously to the right side and epigastrium. The acid vomiting and spasm are often greatly relieved, as shown by Dr. Prout,[1] by the use of frequent draughts of warm water, containing one or two drachms of carbonate of soda to the pint. The first portions are generally rejected, but the fluid is afterwards retained, and the pain and retching abate. Laudanum may be combined with it. Emetics should not be given; they are likely to add to the risk without helping the stone to pass through the ducts. After the stone has passed the common duct, it is better to leave its expulsion to nature than to give purgatives; the less the bowel is irritated, the better the prospect of the stone passing. If there be decided tenderness in the hypochondrium, leeches afford relief.

Medicines have been prescribed with the view of dissolving the calculi in the gall bladder. Although at one time they were in repute, it is not easy to see how some of them, such as ether, chloroform, and turpentine, can act; indeed it is probable that the only benefit they afford is by quieting spasms.

Alkaline medicines have been recommended with better reason, but it is doubtful whether they have a solvent effect upon the stones. If the bile would be rendered more alkaline, there might be less chance of deposition, and possibly some diminution of the calculi take place, but we are in the dark as to the effects of such remedies. The alkaline medicines recommended are, carbonate of soda and phosphate of potash. Their use should be maintained for some time. The mineral waters of Carlsbad, Vichy, Ems, and Marienbad have great repute in cases of biliary calculi. Frerichs says that they have certainly proved efficacious remedies against gall-stones. Dufresne recommends Vichy, and for delicate persons, Ems. There is no good evidence, however, that these waters have any solvent effect upon the stones, and though patients are relieved there by passing the stones, it is not possible to say that they might not get rid of them elsewhere. Frerichs thinks that the quantity of water taken produces a more copious flow of bile, and hence the calculi are forced onwards

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[1] *Nature and Treatment of Stomach and Renal Disease,* 4th edit. p. 257.

more easily. As most of these waters can now be had imported, it would be well to try them in England. The patient's diet should be regulated. He should avoid fat substances and malt liquor. He should be enjoined to take moderate exercise, and to be in the open air as much as possible. The visits to the watering-places above mentioned have the advantage of combining fresh air and pleasant scenery, all of which improve the general health, and it may be presumed that the better this is, the more chance there is of the bile being kept in a normal state.

SUPPURATIVE INFLAMMATION OF THE LIVER.

By W. C. MACLEAN, M.D.

DEFINITION.—Diffuse or circumscribed inflammation of the paren-
chyma of the gland, resulting in resolution or in suppuration, the ab-
scesses in the latter case being sometimes single, sometimes multiple.
A disease for the most part confined to hot and malarious countries,
rarely seen before the age of 20 or after 45, often associated with
dysentery, which it seriously complicates.   Occurring sometimes
in an insidious form, unmarked by very distinctive symptoms
or much constitutional disturbance; or, of a more acute type, with
local and sympathetic pain, pyrexia, and other symptoms depend-
ing on the situation and extent of the inflammation.   The duration of
the disease cannot be defined, varying as it does from a few days, when
it may be presumed resolution has taken place, to 120 or more, after
suppuration.

SYNONYMS.—Hepatitis Acuta; Hepatite; Inflammation du foie, *Fr.*;
Leberentzündung, *Germ.*
In treating of the disease which is to form the subject of this article,
I prefer the term "suppurative inflammation of the liver" to that of
hepatitis; for inflammation of the capsule, *perihepatitis*, and inflamma-
tion of the parenchyma, are often both included under the latter term;
and these diseases, although alike in some of their symptoms, differ in
their site, termination, and consequences. The term Hepatitis Chronica
is used in a still more extended sense, for, particularly in military prac-
tice, it is frequently applied to diseases which have no etiological relation
to one another, and in which inflammatory action has in fact played no
part from first to last. This confusion results in part, from the necessity
of conforming to the official classification of diseases in use in the
British and Indian armies.

ETIOLOGY.—Suppurative inflammation of the liver is a rare disease
in temperate climates. When pus is found in the liver in tem-
perate countries, unless the case has been imported from a hot
climate, the abscess can generally be traced either to inflammation
following a direct injury, such as a blow in the right hypochondrium,
to the existence of septic matter in some distant part of the body, or
to inflammation of branches of the portal system after operations about
the rectum.   In those rare instances in which foreign bodies become

lodged in the liver, abscesses may form around them, and the s:
result follows the intrusion of lumbrici from the intestine along
biliary ducts into the substance of the gland. Only one exampl
hepatic abscess following a blow has come under my observation.
soldier of intemperate habits, while intoxicated, was run away with
a horse and dashed with violence against a gate-post; an enorm
abscess of the liver followed, which proved fatal. Instances of
deposits in the liver following suppuration in distant parts of
body, are related by many surgeons; and Budd and Frerichs
the writings of Dance, Cruveilhier, and Dr. Jackson of Calcutta,
examples of hepatic abscesses following phlebitis of the portal
after cauterization of a cancer of the rectum, operations for fistula in
the extirpation of hæmorrhoids, and violence in reducing prolaps
the rectum. This is anatomically intelligible, when it is rememb
that the pelvic veins of the hæmorrhoidal plexus communicate
the portal system through the inferior mesenteric vein.

In the pathological museum of the Army Medical Departmer
Netley, there is a preparation of the liver of a man who die
phthisis, showing a cavity, the size of a walnut, in the lower pa
the right lobe, in which a needle two and a half inches long, of a
colour, was found. The patient had swallowed the needle two y
before. The cavity of the abscess communicated with the duode
immediately below the pyloric orifice of the stomach; through
opening the pus had drained away unobserved. The existence of
abscess does not appear to have been suspected during life.

In the same collection there is a specimen showing the liver
forated in every direction by lumbrici, around which small absce
had formed. The gall-bladder and biliary ducts are also dister
with these filthy intruders, and some were found in the stomach
duodenum, in the nares also, and even in the frontal sinuses, prob
sent there in the act of vomiting. The specimen was taken from
body of a Maltese boy, two years of age, who died of dysentery;
whether or not symptoms of hepatic mischief were noted during li
not recorded in the brief history of the case. Suppurative in
mation of the liver is a disease of hot and malarious countries,
for reasons not yet well known, not equally prevalent in all.
it is common and fatal in India, while in the West Indies
disease is rare, and the mortality from it is trifling. In both c
tries the temperature is high, and as regards the use and abus
alcoholic liquors, the habits of Englishmen are much the same in
Dr. Parkes thinks that the greater use of highly spiced food and
skilful cooking among soldiers in India may to some extent exp
the difference. But I suspect our countrymen in the West Indie
nearly as much given to stimulate their languid appetites by hot
pers as Anglo-Indians. It is more probable that the insular cli
exercises a salutary influence. There is no lack of malaria in the V
Indies, but it is less generally diffused—more local than in India;
although the temperature there is high, the air is tempered and pur
by the regular action of the sea-breeze in a manner quite unknow

dia, save over a limited area of that vast continent contiguous to the
. In the West Indies, Europeans can, and do, expose themselves to
 sun with comparative impunity. English soldiers undergoing penal
cipline labour in the quarries and on the roads during the hottest
rt of the day maintain good health; but on the continent of India,
similarly employed, they would certainly suffer severely from sun-
oke, hepatic and other affections of an acute type.

It is impossible to overlook the influence of continued high tempera-
re in causing suppurative inflammation of the liver, although some
eemed authors have made light of it. A most insidious and fatal
m of the disease prevails on the continent of India in those parts of
 country where great heat and concentrated malaria act together.
orehead,—while he admits that heat is under certain conditions a
ect exciting cause of inflammation of the liver, and explains in this
y, "its occurrence in the hot months of the year in plethoric
ropeans lately arrived in India, with excreting functions deranged
 free living,"—regards "external cold acting on systems depraved by
 cachexia induced by residence in the tropics, as the most frequent
use of hepatic inflammation." Sir Ranald Martin also insists much
 this view, having often seen acute inflammation of the liver follow
posure to the influence of a cold north wind on people issuing from
ated ball-rooms in Calcutta.

Intemperance in drinking exerts a powerful influence in predispos-
g to this disease. It is well known that spirit-drinking is an exciting
use of inflammation of the connective tissue of the gland, yet at
etley, where we see all the invalids of the British army from India,
rrhosis is by no means a very common disease. I believe the explana-
on to be that the intemperate there are, most of them, cut off by more
ute diseases, such as delirium tremens, acute inflammation of the
er, and dysentery, before there is time for the development of so
ronic an affection as cirrhosis.

According to Waring's invaluable statistical inquiry into the patho-
gy of abscess of the liver, out of forty cases in which the habits of the
ferers were noted, 67·5 per cent. were intemperate.

Indolence and excess in eating, combined with heat and malaria,
 probably the most active causes of the disease amongst men and
men in the higher classes not given to intemperance in drinking.

The frequent association of hepatic abscess with dysentery has been
ady briefly adverted to in the article "Dysentery" in the first volume
this work. The opinion that suppuration in the liver is most fre-
ntly caused by ulceration of the intestines, the stomach, the gall-
dder, or gall-ducts, is one that, although held to some extent by
er authors, has found its ablest advocate in Dr. Budd. The theory
hat the liver becomes involved by some contamination of the portal
d. Dr. Budd holds that small and scattered abscesses result from
tamination of the portal blood, either by pus, formed by suppurative
ammation of one of the small intestinal veins, or by matter of other
d resulting from softening of the tissues; and when the inflamma-

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tion has been diffuse, ending in a large collection of pus, he attributes the result to the absorption of the fetid gaseous and liquid contents of the large intestine in dysentery, conveyed immediately to the liver.

I have already stated (article "Dysentery," vol. i.) that in dysentery of long standing it is a rare thing to see a sound liver; a careful examination of the post-mortem registers at Netley has satisfied me that this observation has even a wider application than I was aware of. Dr. Budd's exclusive theory has not been generally accepted by physicians who have investigated it with care; in India, in particular, it has few supporters. I can only very briefly indicate the facts that militate against it. While the frequent co-existence of the two diseases is admitted, if Dr. Budd's explanation were true, abscess of the liver ought to be a much more common disease than it is. Thus, as has been noticed by Waring and many other writers, it does not follow ulceration of the glandular structures in the intestine in enteric fever and other affections.

Although dysentery is occasionally seen in temperate climates with ulceration, softening, and even gangrene of the mucous tissues, hepatic abscess is extremely rare. Thus, in Dr. Baly's oft-quoted cases in the Milbank Penitentiary, amounting to "many hundreds," hepatic abscess was not found in a single case.

In the Pathological Museum at Netley there are forty-eight specimens of abscesses of the liver. In thirty-four the abscess was uncomplicated with any intestinal lesion; in nine there was a dysenteric history, but no record of any intestinal lesion; in five, hepatic abscess and intestinal lesions co-existed. In three of the above it is specially noted that the dysenteric symptoms followed the formation of abscess.

In fifty of Morehead's fatal cases of dysentery there was no abscess of the liver, and in twenty-one of his fatal cases of abscess there was no ulceration of the intestines. Dr. Parkes examined twenty-three fatal cases of dysentery in India and Burmah, and found consecutive hepatic abscess in five, or in 21·74 per cent. Mr. Waring collected from six different regiments serving in the Madras Presidency 260 fatal cases of acute dysentery; there were sixty-eight complicated with abscess, or 26·15 per cent. The same author gives 300 fatal cases of abscess of the liver from Indian records; in only 27 per cent. was the hepatitis preceded by dysentery. In 204 of the cases where the condition of the large intestine was noted after death, there were no appearances of dysentery in fifty-one. Out of twenty-five fatal cases of dysentery in the post-mortem register at Netley, mostly from India, abscess of the liver was found in only three.

It would be easy to multiply evidence of the same kind; enough has been given to disprove the exclusive pyæmic theory. But I am far from denying that it accounts in a reasonable way for an uncertain but probably considerable number of cases of suppurative hepatitis resulting in multiple abscesses.

MORBID ANATOMY.—It is not often that an opportunity is afforded of examining the liver before the formation of pus has been completed.

In a few cases of multiple abscesses I have seen the process in its different stages going on in different parts of the same liver. Allowance being made for peculiarities in the structure of the affected organ, there is, as Morehead has observed, no difference between the formation of an abscess in the liver and the same process elsewhere.

There is first a stage of hyperæmia, of turgescence, circumscribed or diffuse, as the case may be; when cut into, the part bleeds freely, and often presents a granular appearance. This granular surface is quite visible in some of our Netley specimens that have been many years in spirits.

Bounding this red and softened portion there is a buff-coloured ring of uncertain magnitude. Sometimes this pale yellow colour is widely diffused, as in a case of mine examined at Netley in July 1867—in which a superficial abscess on the convex part of the right lobe had discharged into the right lung, the buff colour pervading the whole of the affected lobe. At a later stage lymph is effused into the congested portion, which now assumes a pale colour; on section, yellowish points containing pus will be seen thickly distributed over the section. These pus spots, according to Frerichs, "first form in the centre of the lobules, the margins still remaining firm." The pus points rapidly coalesce, forming an abscess, large or small, according to the extent of the inflammation and the amount of effused lymph.

The condition of the hepatic tissue surrounding the abscess varies. Sometimes it is seen to be of a dark colour, and hardened; in others this dark colour gradually fades into the pale yellow described above; and again, the boundary between the two is sharply traced.[1]

The condition of the gall-bladder is, as might be expected, variable, depending on the duration of the case, the amount of hepatic tissue implicated, and other causes affecting the quality of the bile. It has never happened to me to see its coats affected with inflammation in connexion with hepatic abscess; but I have noticed a great variety of conditions in the bile found in it after death. In some it is black, thick, and tenacious; in others grass-green and viscid; and in a few cases thin, almost watery, and of a brownish-red appearance. Where inflammation is found it has probably been excited by the vitiated nature of the biliary secretion.

[1] In the *British Medical Journal* of June 22d, 1867, there is the notice of a paper read before the Berlin Medical Society by Dr. Cohnheim, detailing the results of his observations on the formation of pus as a product of inflammatory action. In this paper Dr. Cohnheim announces, as the result of his observations and experiments, that pus-corpuscles not only closely resemble white-blood cells, as has long been known, but are identical. "He has," says the able correspondent of the Journal, "proved that pus-corpuscles are actually white cells which have emigrated from the blood-stream." If this view of pyogenesis should be established, the old theory "which refers the origin of pus-corpuscles to the proliferation of cells or germinal matter in connective tissue, has received its death-blow." It appears to me also that it will throw a new light on the formation of purulent collections in the liver in that obscure class of cases where such abscesses are found without any of the recognised signs of inflammatory action in men and women suffering from malarial cachexia, in whose blood white cells abnormally abound. If the new view is accepted by pathologists generally, it ought to give "a death-blow" to something more than a theory of pus formation; it seems to explain also why blood-letting fails so often in this class of cases to prevent it.

Kind, Position, and Number of Abscesses.—The abscesses resulting from the suppurative process may be single or multiple, superficial or deep-seated, circumscribed or diffuse, encysted, or limited merely by an ill-defined and incomplete lymphy deposit.

Some abscesses are circumscribed by a boundary of effused lymph, more or less complete; others are quite undefined, with their margins ragged and shreddy. A few are enclosed in cysts of varying thickness. Only seven out of forty-eight cases in the Netley Museum have distinct well-defined cysts of varying thickness. In two specimens the investing membrane has attained to a "cartilaginous thickness," and in another an inverted cyst is shown nearly as thick as ordinary wash-leather. These membranous investments are evidently formed by the consolidation and organization of the circumscribed effused lymph, assuming the form of condensed connective tissue. Some cysts are perfectly smooth within, while others are more or less granular. Abscesses of old standing have their internal surfaces smooth; those of recent formation are more or less ragged, shreds of lymph and softened hepatic tissue hanging from the walls of the cavity. Abscesses may form in any part of the gland. In Mr. Waring's collected cases, the right lobe alone was affected in 163, or 67·355 per cent.; the left lobe in sixteen, or 6·611 per cent.; and both lobes in thirty-five, or 14·462 per cent. In the Netley preparations the abscess is situated on the convex and upper and outer part of the right lobe in thirty-six cases; on its concave surface in three; while the left lobe is affected in only seven cases; in one the abscess is intermediate; both lobes were affected in five of the cases.

Abscesses vary infinitely in number. Dr. Parkes mentions a case in which ninety were found, and in Mr. Waring's collected cases the number varied from one to thirty-six. In the Netley collection thirty-seven are single and eleven multiple. In three the abscesses are said to have been "numerous;" but as only portions of the liver are preserved, it is impossible to say how many there were; the rest vary from one to six, and in one case the whole organ is converted into the sac of a huge abscess, which contained seventeen pints of pus, and two of a thin serous fluid.

Quantity and Quality of Pus.—The quantity of pus varies from an ounce or two to the enormous quantity recorded above, viz. seventeen pints. In another of the Netley cases seven pints and a-half were found, the abscess involving the whole of the right lobe. In Mr. Waring's collected cases the quantity varied from four ounces to a gallon, which large amount was found in two cases.

Pus has been observed in hepatic abscesses to present every variety of colour, consistence, and odour. According to my experience, as a rule, it is "laudable," that is, of the consistence, colour, and slight odour of a so-called healthy abscess. I have seen examples presenting the red and pinky colour described by various authors, and others in which the pus had a marbled appearance, given to it by streaks of a pinkish colour, composed of blood intimately incorporated with pus. Much depends upon the circumstances of each particular case, whether

or not air. for example, has had free access to the suppurating cavity, as after surgical operations for the relief of abscesses, in which case the pus will generally soon lose its " laudable " character, and become more or less offensive. Mr. Waring has noted it as " thick," " creamy," 'white," " greenish," " sero-purulent," " brownish," or " reddish," " dark yellow," " very offensive matter mixed with sloughy shreds," and so on. Examination with the microscope will often reveal portions of hepatic tissue, and where the abscess has found its route of discharge through the right lung, traces of lung tissue may also sometimes be found.

Modes of Discharge.—Abscesses of the liver may remain intact until death, or may find issue (*a*) externally through the abdominal walls, (*b*) through the diaphragm into the lung or pleura, (*c*) into the pericardium, (*d*) into the stomach, (*e*) into the intestines (duodenum or colon), (*f*) into the abdominal cavity, (*i*) into a pouch of peritoneum, (*j*) into the hepatic vein.

Of the Netley Museum cases, twenty-nine remained intact at death, some tending towards the outer walls, others to the diaphragm ; two were discharged by operation, six opened into the right lung, three into the cavity of the chest, three into the pericardium, two into the stomach, one into the duodenum, one into the colon, and one into a pouch of peritoneum. In Mr. Waring's oft-quoted three hundred cases,[1] the abscesses terminated in the following manner :—

	No.	Per cent.
Remained intact	169	56·335
Evacuated by operation, a solitary abscess being present	29	
„ „ there being numerous abscesses, one opened, and the others remaining intact . . .	18	16·000
One abscess opened by operation, another subsequently bursting into the abdominal cavity	1	
Opened spontaneously into the thoracic cavity	14	4·666
„ „ into the right lung	28	9·333
„ „ into the abdominal cavity	15	5·000
„ „ into the colon or large intestines	7	2·333
„ „ into the stomach	1	
„ „ into the hepatic vein, leading to the vena cava	2	
„ „ into the hepatic vein, at its junction with the vena cava, and another communicating with the cellular tissue around the right kidney	1	
Communicated with the hepatic ducts	1	
„ with the right kidney	2	
„ with the gall-bladder	1	
„ with an abscess in the iliac region	1	
Opened spontaneously through the ribs in the back	1	6·333
One abscess had opened into the colon, and another had passed off by the hepatic ducts into the duodenum	1	
One abscess had opened into the stomach, a second into the duodenum, and a third had been evacuated by operation	1	
One abscess had opened into the abdominal cavity, and a second into the lungs	1	
Terminated in erysipelas of the lower extremities, simulating phlegmasia dolens, the abscess opening into the lungs	1	
Doubtful	5	
	300	100·000

[1] Waring's Inquiry into the Statistics and Pathology of some points connected with Abscess in the Liver.

Do abscesses of the liver ever undergo absorption ?

Nothing is more common than to observe puckered depressed cicatrices on the surface of the gland. The Netley collection contains many such specimens. In the early years of my Indian service these were invariably regarded as evidence that a superficial abscess had formed and been absorbed in that spot. That some of these admit of such an explanation can hardly be doubted, but more recent and accurate observation has demonstrated the frequent association of these cicatrix-like depressions with the syphilitic dyscrasia ; and where they contain the gummatose nodules, the ' greyish-yellow fibroid nodules ' of Frerichs, with palpable evidence, in other organs, of constitutional syphilis, no doubt can be entertained as to their true nature.

Dr. Morehead gives three cases in which the process of absorption had certainly taken place, and one in which recovery in this way was probable. In the cases where absorption had evidently occurred, a " putty-like substance " alone remained in the sac, identical, I presume, with the " cheesy matter " described as found under like circumstances by Frerichs and other observers.

CLINICAL HISTORY AND SYMPTOMS.—There is no disease so difficult to describe as suppuration of the liver, and, paradoxical as the saying may be, I suspect this difficulty is experienced most by those who have had most experience in dealing with it. The task assigned to me in this article is to describe suppurative inflammation of the liver, but, as I have already said, there is a disease so closely resembling it in many of its symptoms, and, consequently, so often confounded with it, that I must briefly advert to it here. This is inflammation of the investing capsule of the liver, the *perihepatitis* of Frerichs and other writers.

Morehead appears to think that this disease is not common in India. It is always with diffidence that I venture to differ from this accurate clinical observer of tropical disease. But I have long held a different opinion, and often given expression to it in my published writings, and in my lectures in the Army Medical School. The diagnosis is far more important than is at first sight apparent. A great deal of the support that is still given to blood-letting in the treatment of suppurative inflammation of the liver is based on the apparent success that often follows the large abstraction of blood, in preventing, as is imagined, the formation of abscess, whereas, in a not inconsiderable number of cases, the measure has been directed against a disease which has no such termination. Thus, a very recent writer, after acknowledging that he recognised the case to be one of capsular inflammation, informs his readers that he bled his patient to the enormous extent of 166 ounces, besides purging him severely by a combination of active cathartics, and, because no abscess followed, draws the conclusion that the bleeding prevented suppuration.

Perihepatitis is met with under two forms, first as an idiopathic disease ; and secondly, consecutive to abscess of the parenchyma.

Omitting those cases in which it occurs as a part of general peritonitis from any cause, and those in which, according to Frerichs, "the capsule becomes inflamed in right pleurisy," the inflammation spreading to the serous covering of the diaphragm and of the liver, the capsule of the gland may inflame just as the pleura does after chills, when a person has been overheated. The symptoms resemble those of pleurisy much more than those of inflammation of the parenchyma. There is pain which is sharper and more acute than in true suppurative inflammation. This pain is sensibly aggravated on pressure, or a full inspiration, or after any movement. It is described often by the common expression " stitch in the side." The liver is not congested, and the urine does not differ from its normal appearance; with the above symptoms there is more or less febrile excitement. The result is exudation of lymph between the gland and its capsule, strong adhesions, sometimes extending to neighbouring organs, and occasionally the presence of a thin layer of purulent matter. In the case of an invalid from India, who died lately at Netley from aneurism of the arch of the aorta, the liver was so closely embraced by its adhering capsule as to cause atrophy of the whole organ. This man had a history of " hepatitis," and had doubtless been treated for inflammation of the parenchyma.

The second form of perihepatitis is that in which it occurs when an abscess of the parenchyma involves the capsule as it makes its way to the surface. The symptoms will then depend upon the position of the abscess. As I shall presently show, it often happens that active treatment for the prevention of suppuration is commenced at the setting in of these symptoms, which indicate, not the commencement, but the last step of the process. I know no point in the whole clinical history of hepatic inflammation of so much importance as the one just described.

The following are examples :— W. M. A——, medical officer of the Madras army, of a delicate constitution, had suffered severely from dysentery, and occasionally passed some muco-purulent matter at stool. This gentleman paid me a visit, and spent some hours with me, in the Medical School at the Residency of Hyderabad in the Deccan. I observed that he looked ill when he arrived; he confessed that for many days he had been ailing without any very marked symptoms beyond loss of appetite, a feeling of general malaise, and a disinclination for any active physical or mental exertion. While engaged in examining some of the pupils, he suddenly experienced a distinct rigor, with a feeling of approaching syncope, followed by a copious perspiration. After he had rested a little in the recumbent position, I drove him to my house. In the evening he rallied sufficiently to return to his own home, four or five miles distant. Next day he resumed his duty, experiencing now and then transient chills followed by slight flushes of heat. In a few days from the date of his visit to me, while playing whist with some friends, he was suddenly seized with acute stabbing pain in the right hypochondrium, above

the false ribs, with sharp fever, followed by cough, and sympathetic pain in the shoulder—in a word, the symptoms usually said to denote "acute hepatitis." Had I seen him then, I certainly should have dis-suaded him from following the treatment his own judgment suggested, viz. active blood-letting, general and local, with purgatives, followed by calomel. I saw him thirty-six hours after the setting in of the above symptoms, free from pain, but much prostrated, and expectora-ting purulent matter, from an abscess of the liver, through the right' lung. The issue of the case was fatal ; and on examination I found an abscess on the convex surface of the liver, communicating with the lung, which was much disorganized, and two other abscesses deep in the right lobe, and extensive ulceration of the great intestine. I never entertained a doubt as to the true nature of this case. The active treatment began when it could avail nothing. The acute symptoms indicated a curative process, namely, adhesive inflammation of the capsule to the diaphragm, preparatory to the advance of an abscess, long antecedently formed, and its evacuation by the lungs. It is im-possible to conceive that the mischief revealed after death dated only from the setting in of these symptoms. I always regarded this as an unequivocal case of hepatic abscess, consecutive on ulceration of the intestines.

Here is another case, with a more fortunate issue : Lieut. ——, H.M. —— regiment, arrived at Madras, from Secunderabad in the Deccan, in charge of a detachment of troops. This officer, twenty-four years of age, had been two years in India, had lived foolishly, drinking to excess, keeping late hours, and frequently exposing himself in the snipe-field, a hot sun overhead, his feet being immersed in water much colder than the surrounding air. For days before I saw him he had been ailing, eating nothing, or next to nothing, but drinking much. His complexion was dark and muddy, his eyes were congested, his breath heavy and alcoholic, he had no fever, no heat of skin, and his pulse did not exceed eighty. He had little to say in the way of complaint, except that he was "ill," and unfit for anything but to recline on a sofa. I examined his liver with care. It was enlarged, without doubt ; he had a sense of fulness, but allowed me to take any liberties I pleased in the way of palpation and percussion. His urine was high-coloured, loaded with lithates, and so turbid that I could not see the bottom of the vessel. I felt convinced that I had to do with deep-seated mischief in this young man's liver; but, even had I been disposed to put active antiphlogistic treatment in force, there was ample reason in his habits to forbid it : a moderate bleeding would have been followed by delirium tremens.

Short of such measures I did what the case admitted of, and gave orders to be called as soon as acute symptoms set in, which I con-fidently looked for. Within fifty hours I was called to him. After a sharp rigor, he had stabbing pain in his right side with fever, cough, a "to and fro" sound at the base of the right lung, soon followed by purulent expectoration. Dr. Paul, of the General Hospital, saw the

case with me; we were quite at one about the treatment. We well knew that antiphlogistics could avail us nothing here, that the mischief had been done before the patient came under medical observation. His strength was therefore sustained by suitable means, pain was allayed by fomentations and opiates, the mineral acids with quinine were given, and in a short time we had the satisfaction of sending this young man to England quite convalescent. Had he been seen for the first time on the setting in of the acute symptoms, and without a knowledge of his antecedents, and treated according to routine, I think the issue would have been different. Blood-letting could not have prevented the formation of an abscess (that was already there), although it would certainly have weakened the patient; and if mercury had been given, and it had acted, as it is supposed to act, viz. by preventing the effusion of lymph, or causing its absorption after effusion, the result would have been equally unfortunate, for adhesion of the opposing surfaces would have been prevented or destroyed, the conservative operations of nature would have been interrupted, and escape of the abscess into the abdomen must have ended the case. Having thus premised, I proceed to consider the

SYMPTOMS OF SUPPURATIVE INFLAMMATION.—These are said to be a sense of fulness and weight in the right hypochondrium, pain, inability to lie on the right side, fever, disturbance of the digestive organs, cough, and sympathetic pain in the shoulder, and, according to some authors, jaundice. In the two cases just given, and I could give many such, it will be seen that most serious and destructive mischief may be going on, and yet the symptoms may be such as to excite little alarm either in the patient or his attendant, unless he be a wary and experienced practitioner. Looking at the above symptoms in their order, it will be found that enlargement of the liver, with the sensations to which it gives rise, will generally be present if a considerable part of the gland is implicated in the morbid process, but every tropical physician will call to mind numerous examples in his experience of abscesses as large as an orange having been found without any enlargement likely to attract attention.

Pain.—The liver is not a sensitive organ, and the most serious mischief may be going on deep in the substance of the parenchyma, and yet no pain may be complained of. On the other hand, pain may be acutely felt, and yet, as I have explained, the parenchyma may not be affected at all. This symptom is always influenced by the depth at which the inflammation is going on, being usually well marked where the inflammation is superficial, less so, or absent, where it is deep. It was present in 85 per cent. of Rouis' cases (Frerichs). When pain and enlargement co-exist, the case is more serious.

Fever.—In latent abscess there may be no disturbance of the circulation, until the abscess approaches the surface. It is astonishing how little the pulse is affected, often not exceeding 80, when a large abscess may be in process of formation deep in the gland. When, however.

the capsule is affected, there is an immediate rise in the frequency and sharpness of the pulse, and in all cases of superficial inflammation it is affected from the first. Careful observations with the thermometer made by me in the clinical wards at Netley, have, in every instance of suppuration of the liver recently under observation, shown a rise in temperature of from one to three degrees Fahr.

Disturbance of the Digestive Organs.—Loss of appetite is an occasional, but by no means invariable, attendant on the disease; as might be expected, it is common in the intemperate, but often there is little disturbance of the digestive system. I can confirm the fact noted by Frerichs, that when suppuration commences, " the tongue becomes covered with a grey or yellowish coat." On the other hand, violent gastric symptoms may be excited when the stomach or duodenum are implicated, as an abscess tends to discharge into one or other of the cavities.

Respiration is disturbed, or not, according to the situation of the inflammatory action. When an abscess is making its way through the diaphragm, a short dry cough is then developed, often preceded, and for a short time attended, by a friction murmur at the base of the lung, which, however, does not last long. Sympathetic pain in the shoulder is much insisted on by most writers, and is said to be most common when the inflammation is on the convex portion of the liver. Annesley and Parkes say this is a symptom of uncertain value, and it is often present in chronic cases where there has been no suppurative inflammation.

The urine gives most valuable information, and should be narrowly examined. Dr. Parkes says, " that in the exquisite forms of hepatitis, as witnessed in hot climates, the urine is most highly febrile, and that the pigment is greatly increased. The few observations which have been made, imperfect as they are, show that there must have been an increase in the urea. When large abscesses have formed in the liver, however, and the functions of great part of the hepatic tissues are abolished, the urea is certainly sometimes deficient. The urine is then copious, pale, non-sedimentous, and non-albuminous." There is a field here for fresh researches, to which the attention of tropical physicians is earnestly invited.

Jaundice, although often given by systematic authors as a symptom of inflammation, is of little or no value for diagnostic purposes. Excepting to a slight degree in a few exceptional cases, I have never seen it. In Morehead's clinical cases it was extremely rare, but it seems to be rather more common in Algeria than in India (Haspel).

It will be seen from the above, that the symptoms of suppurative inflammation of the liver cannot be said to be very distinctive, yet, I venture to say, that a wary physician will not often be deceived. In the first place, practitioners, in regions where this disease may be looked for, should always examine the condition of this great gland, even where there may not be much to call attention to it; if this be done, although little value can be attached to any one symptom, it is hardly

possible by a careful attention to the patient's past habits and history, **to his general** condition, and to the result of a careful examination of **the organ with all** the aid to be derived from percussion and palpation **and the use** of the thermometer, to miss coming to a conclusion on **which to base** rational treatment.

Remembering also how frequently abscess of the liver is found in **dysentery, the** physician will, in treating that disease, have a watchful **eye on the** condition of this gland, both as to function and physical **state; and** should dysentery or obstinate diarrhœa follow hepatic **symptoms,** however obscure, and resist rational treatment, no prudent **physician** will fail to suspect hepatic disease. To sum up. In young **sthenic subjects,** who have not been long in the tropics, who have been **living freely,** exposing themselves to the sun by day and to chills at **night, we** may expect to see many of the above symptoms well marked. **Again in** hot low-lying malarial localities, and particularly in men **or women** whose constitutions have been impaired by long residence **in a hot climate,** the symptoms will be more "silent" and insidious, **and in all,** the position of the affected part, as regards nearness to the **surface** or the reverse, the extent of the tissue implicated, and the **contiguity** of neighbouring organs, will exercise a marked influence **on the symptoms.**

Signs of Suppuration.—How can we tell that suppuration has **taken place?** The signs are local and constitutional. Where an **abscess is** situated on the outer aspect of the liver, fulness will be per- **ceptible, and** unless the pus be deep-seated, fluctuation will be detected **by careful and** delicate manipulation. These signs absent, the sudden **setting in** of sharp pain, aggravated by pressure or movement with a **dry cough,** and it may be a to and fro sound at the base of the lung, will **at once raise** the suspicion of abscess on the convex portion of the right **lobe, "** pointing" towards the chest. Deep-seated pain, less acute than **the above,** and attended with vomiting, points to the possibility of an **abscess** pressing for discharge into the stomach or duodenum, and irri- **tating diarrhœa** often precedes its discharge into the colon. There is **a therapeutic** sign of much value in dysenteric cases, it is insisted on by **Morehead,** viz. great intolerance of ipecacuanha. I have verified this **important** clinical fact on many occasions. Chills followed by hectic **fever and** exhausting sweats are signs of much value, but are often **wanting.** How is it that sometimes an abscess no bigger than a small **orange,** deep in the substance of the liver, may set up irritation **enough** to extinguish life, while, in other cases, abscesses twice or **three times** the size may exist for a long time, and cause little or no **constitutional** disturbance? Setting aside cases where the presence or **absence** of dysentery or extreme malarial or other cachexiæ may be **the cause,** the explanation is the presence or absence of a stout cyst. **There is** hardly a day of my life that I have not occasion to point out **to the** gentlemen candidates studying at Netley, not only the absence **in some** cases of chronic phthisis of constitutional irritation, where **there is** evidence of a tubercular deposit having taken place into the

apex of one or both lungs, but also the wonderful comparative restoration to health, as evidenced by increased appetite and weight, cessation of cough, and so on; the explanation being a cretified condition of the tubercular deposit, or its being, cut off by a plastic partition, in either case ceasing to be a source of constitutional irritation. In like manner, where hepatic abscesses are stoutly encysted they may remain for months, perhaps for years, latent, producing little or no constitutional disturbance. In the pathological museum at Netley there are two preparations which will illustrate this observation. A sergeant, invalided from India for chronic hepatitis, presented himself at Fort Pitt. He had to all appearance recovered so completely on his voyage from India, that he was sent to the Depôt for duty. Weeks afterwards, while straining at stool, "something gave way," and an hepatic abscess burst into the pericardium. In this case the cyst was of great thickness, and even of cartilaginous hardness.

In another case the patient, a native of Bombay, had worked on a plantation at the Mauritius; he walked seven or eight miles in search of a fresh engagement, his health being apparently good. "Suddenly he complained of a pain at the pit of the stomach," and died in a few hours: an abscess of the liver burst into the pericardium; the abscess was lined with "a firm cartilaginous membrane." Here were two men who for months, perhaps for years, were able to go about, one of them to labour on a sugar plantation, yet both had abscesses in their livers; the only reasonable explanation of the absence of constitutional irritation being the firm barrier afforded by the investing cysts.

PROGNOSIS.—Abscess of the liver is at best a dangerous disease, and under all circumstances demands a cautious prognosis. When it occurs consecutive to dysentery it is a formidable complication, and the prognosis is unfavourable.

An important element in the prognosis is the point of discharge. Abscesses bursting into the pericardium or peritoneum are always fatal, and that quickly. As a rule, the issue is seldom fortunate when the discharge is into the cavity of the pleura, or where the abscess points externally through an intercostal space. It is much more favourable when it points at the ensiform cartilage. In my experience the largest number of recoveries follow discharge through the lung, and next to that into the intestine. When air obtains free access to the cavity of an hepatic abscess, a favourable result seldom follows, and, whatever be the position of the abscess, the occurrence of much hectic, extreme emaciation, or diarrhœa, is of evil omen. The fact of the abscess being multiple or single must exert a powerful influence on the issue of the case; but it is seldom possible to do more than guess at the condition of the gland in this respect. Where, however, after the evacuation of an abscess (*e. g.* through the right lung or the intestine), a patient does not improve, but continues to suffer from hectic and to emaciate, it is nearly certain that other abscesses exist, and the prognosis must consequently be unfavourable.

DIAGNOSIS.—The diagnosis between suppurative inflammation and perihepatitis has already been given. It is hardly possible to mistake a hydatid cyst for an abscess in the liver, if we attend to the previous history of the case, the absence of constitutional symptoms, the slow development of the tumour, its painlessness, and its smooth and globular shape. If suppuration has occurred, the diagnosis may not be so easy ; even then the past history of the case, if carefully investigated, will lead to a right diagnosis, and, suppuration once established, the principles of management are much the same in both. If the question of abscess or no abscess in a case of dysentery is presented for solution, no physical signs of its existence being present, we may strongly suspect the occurrence of suppuration, when the signs of constitutional irritation are more marked than the severity of the primary disease explains, when nutrition fails, when the patient emaciates and grows hectic, even though there may be no hepatic tenderness or sympathetic pain in the shoulder, or any visible enlargement and fluctuation.

TREATMENT.—Before entering on a course of treatment, more particularly if active treatment be contemplated, it is above all things necessary to look narrowly into the patient's history, and all the particulars of the case, lest we fall into the serious error, already so often mentioned in this article, of mistaking the end for the beginning of the case, and directing treatment to prevent what has already occurred.

In a so-called inflammatory disease, the first remedial measure to be discussed is, of course, blood-letting. It is not necessary to waste words in condemning spoliative treatment in the class of cases occurring in low-lying, hot, and malarious localities, in the persons of those whose constitutions have been impaired by long residence in such places. If anything was wanting to complete the evidence of the utter unsuitableness of this treatment in such cases, the researches of Dr. Cohnheim, on the identity of the white blood-cells with pus corpuscles, already referred to, appear to have furnished it.

The question of the necessity, or otherwise, of bleeding is more likely to arise, when the symptoms of " acute hepatitis " occur in young men with constitutions but little impaired by residence in the tropics. In such, more particularly where the inflamed part of the liver is near the surface, the symptoms are sure to be much more marked and acute than in other forms of suppuration of the gland. The propriety of bleeding in such cases can be supported by a cloud of witnesses, many of them men of great weight and reputation as tropical physicians. But, as in other diseases so in this, the necessity of the measure is called in question. It was my fortune, early in my career, to serve under one who believed in the efficacy of this measure, and, so believing, used it with conscientious perseverance and without hesitation or misgiving. I narrowly watched the results, and, of this I am sure, I saw more cases of suppuration of the liver under this gentleman than I have ever seen since. I was never satisfied that it answered the

end in view, viz. the prevention of suppuration, in a single instance; I have seen it relieve pain, abate fever, and at once reduce the force and frequency of the pulse. On the other hand, the signs of suppuration often followed so quickly as to suggest one of two conclusions, either that pus had formed before the bleedings were practised, or that its formation had been hastened by the proceeding. Where no such untoward occurrence followed, the effect was often to induce, unmistakeably, prostration, anæmia, a long stay in hospital, and protracted convalescence. I have long abandoned the proceeding : in a few exceptional cases I have applied a few leeches to the side or to verge of the anus, but for more than twelve years before ending my Indian career I never once used the lancet.

MERCURY.—Faith in calomel may be said at one time to have attained in India to the dignity of a dogma. It was supposed to exercise a controlling power over hepatic inflammation, preventing suppuration, either by arresting the effusion of lymph, or promoting its absorption after effusion. I have had large opportunities of seeing this remedy tried by men who were, in their time, deemed to be exceptionally skilful in its use. I was never satisfied that any useful result followed its exhibition, other than could be explained by its purgative effects. I have seen it fail so often to arrest suppuration, that I utterly disbelieve in any such power. I have again and again seen suppuration of the liver occur when the patient was actually salivated; going, as it usually did, hand in hand with bleeding, it powerfully aided the malarial cachexy in blanching the patient, depraving the blood, and protracting the period of convalescence. Fourteen years ago I published a paper in the Indian "Annals of Medicine" to the above effect, and the result of my subsequent experience and observation has satisfied me more and more of the correctness of the views then given. In Mr. Waring's short but pregnant chapter on Treatment, appended to his "Inquiry," he gives ample evidence that at best it is "of doubtful utility," and that "its free exhibition is undoubtedly no preventative of hepatic abscess."

The high authority of Morehead can be quoted to the same effect and Deputy-Inspector General Massy, formerly of the 2d Dragoon Guards, in an admirable paper on Hepatic Disease, published in the "Statistical, Sanitary, and Medical Report of the Army, for 1863," vol. v., has added his evidence to that of other recent observers against its use, on the ground that it "deteriorates the health, not unfrequently permanently." Before this physician ever set foot in India, he tells us that, while serving in Chatham, where he saw the invalids from India, he was often led to meditate over them, "uncertain whether mercury or disease of the liver had done most to deprave their constitutions." Year by year, I rejoice to say, the evidence of excessive mercurialization, among invalids from India, is becoming more rare.

Bleeding and the administration of mercury being thus objectionable, what remains ? I answer, Ipecacuanha. For years past, in my

lectures at Netley, I have urged the free use of this invaluable remedy, not only in dysentery, but in suppurative inflammation of the liver. I give it in the same large and efficient doses as in dysentery—from 20 to 25 grains, and even more—and, so far as my experience extends, I am of opinion that it is nearly as efficacious in this disease as in tropical dysentery; the *modus operandi* being the same in both. I am happy to be able to add the valuable evidence of Dr. Massy to the same effect. Dr. Massy was led to use it in acute hepatitis from observing its effects on the liver when he gave it in acute dysentery complicated with congestion of the gland. In Dr. Massy's opinion it "quieted and equalized the circulation," and it certainly "caused nausea, profuse diaphoresis, and frequently large bilious motions." Dr. Massy aided the above effects by the use of leeches—I am far from saying that leeches are never required, but I am persuaded their use ought to be restricted to decidedly acute cases, in the persons of patients with unimpaired constitutions. Sir Ranald Martin has some excellent observations directed against indiscriminate leeching, and points to the enormous quantity of blood lost by their too free and frequent application.

The mode of giving the remedy is the same as in dysentery (*vide* Vol. I. article "Dysentery"), the doses being repeated at intervals of five, six, or eight hours, according to the severity of the case. The side should be freely stuped and fomented, and if the ipecacuanha fails to produce free evacuations, aperient medicine should be given; but, remembering how close is the relation between dysentery and suppuration of the liver, care should be taken in the selection of the purgative. I have, in the article "Dysentery," Vol. I., referred to the fact that Indian observers have noted, since ipecacuanha has come into general use in the treatment of dysentery, that abscess of the liver has been less frequent in their experience. Dr. Massy, in his paper already referred to, gives the following additional testimony to the efficacy of the treatment of hepatic inflammation by ipecacuanha. "My friend Dr. Laing, of the 23rd Royal Welsh Fusiliers, informs me that he uses no other medicine in this disease, and that his practice is remarkably successful, far more so than when he was in the habit of employing mercury, which drug he has latterly altogether discarded." Closely allied to the above method is that recommended by Cutcliffe, Civil Surgeon of Meerut, published in the Indian *Lancet*, of February 15, 1860, and quoted by Dr. Massy. It consists in the administration of tartar-emetic in combination with nitrate of potass, two grains of the former to two drachms of the latter. This is divided into eight powders, one being given every half-hour until pain is relieved, aided, in severe cases, by leeching and fomentations. Dr. Massy, from a trial of this method extending over a year, speaks very favourably of it. I have often used tartar-emetic in acute cases in strong young men, in minute doses quickly repeated, sometimes aiding its effects by small doses of tincture of

aconite,[1] cautiously given; but the combination with nitre seems worthy of an extended trial in the class of cases above described, in which alone I should conceive it to be applicable. Antimonial medicines, if used with the requisite precautions, unlike mercury, leave no sting behind them. If the above means be carefully used, and used early, the symptoms usually described as those of chronic hepatitis will seldom follow; if any dull, undefined uneasiness remains in the side, it will often be dissipated by a blister, the use of the nitro-muriatic acid, and a little quinine. Even should the above treatment fail and suppuration ensue, the patient will be in a much better condition to bear the trying process and its consequences, with his powers unimpaired by depletion and the depraving action of mercury on his blood.

An abscess having formed, is it to be allowed to take its natural course, or is the aid of the surgeon to be called in to evacuate it by puncture? This is a most momentous question for the patient. Some practitioners are so much impressed with the necessity of early evacuation of abscesses of the liver, that they advocate a diligent search for the purulent collection, by what is called "exploration," as soon as they are satisfied, by the presence of certain constitutional signs, that suppuration has been established. I am opposed to this practice both on theoretical and practical grounds, and I may add that it has never been sanctioned by physicians of authority and experience in India. Morehead reprobates it in unequivocal terms. I think it contrary to sound surgical principles, and, as I have elsewhere explained (vide *Lancet*, vol. xxi. 1865, and " Army Statistical, Sanitary, and Medical Reports," vol. vii.), an interference with one of the most conservative operations of the system. If it be true, as I believe it is, that the most favourable route an abscess of the liver can take is through the right lung, and the least favourable—the one giving the smallest number of recoveries—is through the abdominal walls, it appears to me unjustifiable to expose the patient by a surgical operation to the risk of admitting air with its mischievous properties into the cavity of an abscess tending to a comparatively safe point of discharge. As far as I am able to judge, the advocates of puncture draw their support of their favourite practice from a small number of successful cases. The question is simplified a good deal when the abscess is visibly tending to the surface, and must ultimately be discharged through the abdominal walls. Is Nature in such a case to be allowed to operate in her own way; or is the surgeon to anticipate her proceedings? Where the abscess is large, where it is obviously causing much constitutional irritation and such symptoms as hectic, distressing cough, vomiting, or diarrhœa, it is impossible to wait, and the patient will usually be clamorous for operation. Nothing, as has been well shown by Dr. Budd, and more particularly by Dr. Lowe of the Madras Army

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[1] This, in all acute inflammations, is a remedy of much value. It may be given in doses of one drop every ten minutes, until six or eight drops have been given; continuing the remedy in the same doses every hour, *closely* watching its effect on the circulation.

(*Madras Quarterly Journal*, vol. vi.), a physician of much experience in dealing with such cases, can be more different from one another than the method of the surgeon on the one hand, and that of Nature on the other. The surgeon discharges the whole contents of the abscess at once, air takes the place of pus, and however great may be the relief, and it is often very great, from the operation, the issue is usually disastrous. · Decomposition of the discharges from the cavity sets in, unhealthy extension of the suppurative process goes on, under which the patient succumbs, or gangrene proceeding from within outwards at the site of the puncture brings the case to an end.[1] This latter occurrence has resulted in nearly all the operations practised by me on Asiatics, and I have seen the same thing happen after similar operations on Europeans. Nature never acts in this way, but, as described by the authors above-named, and often witnessed by myself, the proceeding is much slower; the pus escapes gradually from several small apertures, the cavity contracting slowly as the pus drains away, while hardly any air finds admission. I have attempted to imitate the process, by valve-like openings, by drawing off only portions of the purulent collection at a time, endeavouring to close the opening with the aid of collodion; I have tried to attain the same end by using a canula furnished with a stopcock but all in vain; sooner or later air entered, and the issue was unfortunate. I have lately, in a paper already alluded to ("Army Medical Reports," vol. vii.), suggested the use of Bowditch's syringe for this purpose. This instrument was constructed for evacuating pus, or fluid of any kind, from the cavity of the pleura without the admission of air. We use it at Netley, and find it answer the purpose perfectly. I have had an opportunity of trying it in one case of abscess of the liver, under the care of Mr. King Sampson of Southampton, with whom I saw the case in consultation. The patient, a young coffee-planter from Southern India, was landed at Southampton in a state of extreme prostration. There was no difficulty in making out a well-defined abscess of the liver, which, although still rather deep-seated, obviously tended to the abdominal walls. It was determined to evacuate the abscess by Bowditch's syringe, which was done; a pint of pus was drawn off, to the great relief of the patient, through an aperture so minute, that the young man's parents could hardly see it, and could with difficulty be persuaded that the pus had come through what appeared to be merely a red point on the skin. No air entered the cavity. The young man sunk in about twenty-four hours after the operation from exhaustion, and possibly from the presence of other abscesses in the liver not accessible. Both Mr. Sampson and myself were satisfied that the instrument, if used sufficiently early in a suitable case, is admirably adapted to secure the end in view—the evacuation of pus

[1] Would carbolic acid, used after the manner of Mr. Lister, be of use in preventing the decomposing action of atmospheric air admitted into such a cavity? It certainly would be worth trying, and there could be no difficulty in washing out the suppurating sac with the mixture of carbolic acid and linseed oil recommended by Mr. Lister, in the proportion, namely, of one part of the former to five of the latter.

without the admission of air into the sac. I hope that practitioners in India will give this method a fair trial, and publish the results.[1]

In puncturing for hepatic abscess, is there any danger of wounding the gall-bladder? I saw this done by one of the most eminent surgeons in France, in one of the hospitals of Paris, more than twenty years ago. Morehead also appears to have heard of cases of this accident. The position of the swelling and its pyriform shape will lead to a correct diagnosis. The patient mentioned above was more deeply jaundiced than I have ever seen any one before or since. When an abscess of the liver is discharging, the utmost care must be taken to sustain the patient by suitable diet, by the use of light unbrandied wine, and, as soon as the case admits of it, by removal to a better climate.

Those who have been most conservative in treatment in the early days of the case will be more successful at this stage than the practitioners who, in their anxiety to prevent suppuration, have been prodigal of their patient's blood and strength.

In conclusion, I cannot avoid repeating an observation often made by me before, in the various discussions that have taken place of late years in the medical press on the subject of puncturing hepatic abscesses. It is this: The invalids from every foreign station garrisoned by the British Army pass through the Royal Victoria Hospital, Netley. Nothing is more common than to see men who have made excellent recoveries after the evacuation of hepatic abscesses through lung or bowel, nothing more rare than to see men with the cicatrix of such an abscess in the right hypochondrium, more particularly in an intercostal space. Where there is an external cicatrix, it is almost invariably situated just below the ensiform cartilage, verifying the *dictum* of Morehead as to the greater frequency of recoveries when abscesses pointing outwardly occur in the thin part of the right or well towards the left lobe.

GANGRENOUS INFLAMMATION OF THE LIVER.

This is a disease of which I know nothing from personal observation, save in the form spoken of in the preceding article, viz. as a sequel of suppurative inflammation from the decomposing action of atmospheric air admitted into the cavity of a hepatic abscess by surgical operation. I am quite aware that a deceptive appearance of gangrene often occurs from the blackening of the tissue in and around an abscess by gaseous emanations from the decomposed discharges. Dr. Budd (Diseases of the Liver) has cautioned us against falling into this error. In every case the gangrene was not confined to the liver, but extended to the tissues around the opening of the abdominal walls.

[1] Since the above was in type, I have used this instrument again and again, and in some instances with the happiest results. In single abscesses tending to the surface, I have no hesitation in saying that it affords the best hope of a happy issue.—*Vide* " Army Medical Reports," vol. ix.

Several cases are mentioned by authors of gangrene of the liver following mortification in some remote part of the body. When this occurs, the affection is never confined to the liver, but is found also in other organs, more especially the lungs and spleen.   Dr. Budd gives the details of a case of this kind, communicated to him by Mr. Busk, in which gangrene of the liver and other internal organs followed mortification of the toes from exposure to cold.   The dead parts were removed, rigor set in, followed by typhoid symptoms, and death on the sixth day.   Gangrene of the liver, lung, and spleen was found after death. Mr. Busk thus describes the appearances in his case: "On the outside of it the liver was not discoloured, and presented no marks of recent inflammation; when it was cut into, numerous ragged cavities of various sizes were found, containing hepatic substance in a state of complete gangrene, and reduced in many of them to a semi-fluid and ash-coloured flocculent matter, separated by a very defined line from the surrounding substance, which, in immediate contact with the gangrenous portions, was of a deep greenish slate colour." (Budd, Diseases of the Liver.)

It is needless to add, that this rare affection is beyond the reach of curative treatment.

CHRONIC ATROPHY OF THE LIVER—CIRRHOSIS.

By Edward Goodeve, M.B.

DESCRIPTION.—Chronic atrophy of the liver occurs in three or four different anatomical conditions. The form most frequently met with, and therefore of the greatest clinical importance, is the disease called Cirrhosis, hobnailed liver, or granular liver. In the present article it is proposed to describe chiefly this form. The course of the disease, and the most important symptoms in all the varieties, are sufficiently alike to make the account of one form answer for all.

Cirrhosis is an insidious chronic disease, often commencing with apparently trivial symptoms, and those chiefly referable to the digestive organs; in other instances, however, beginning with hepatic pain and enlargement; proceeding in both cases slowly, sometimes very slowly, to considerable or extreme atrophy of the liver, with exhaustion, anæmia, ascites, and death.

The symptoms of the disease will probably be best understood by a study of the anatomical alterations which the liver and other organs undergo.

PATHOLOGY.—The first most obvious and striking change in the advanced stages of Cirrhosis is found in the size of the liver. This is greatly diminished—often to half its bulk; it presents a curious tuberculated or granular surface, the resemblance of which to the hobnails of shoes has obtained for it the name of hobnailed liver. From the yellow or yellowish colour of parts of the surface of sections Laennec gave it the name of Cirrhosis.

The atrophied, *cirrhosed*, or contracted liver has undergone considerable changes throughout its structure. Externally it is of greyish-drab or whitish colour : it is reduced to two-thirds or one-half its natural size; many specimens not exceeding 1 lb. 8 oz. or 2 lbs. in weight. The diminution is pretty uniform in most of the dimensions: the edges waste first, and are often reduced to a thin, almost membranous condition, with the nodular or tuberculated state existing where there are remains of parenchyma. The left lobe is usually more atrophied than the right; indeed, often appearing as a mere thin expansion attached to the right lobe. The thickness of the liver is not diminished in the same proportion as the other dimensions, so that as the organ is chiefly reduced in length and breadth, the remainder

assumes a somewhat rounded or globular shape. The consistence is firm, tough, or leathery; the hardness is especially noted in the earlier stages of the disease. The capsule is generally opaque, adherent to the subjacent parenchyma; and, if there has been peri-hepatitis, it may be much thickened, and covered with false membranes or adhesions both on its upper and under surfaces, which may thereby be attached to the neighbouring parts. In some cases the adhesions have lengthened out, as if by traction of a diminishing succeeding an enlarged liver. Both the superior and inferior surfaces are studded with semi-globular, sessile, warty prominences, varying in size from a pin's head to a quarter or half an inch in diameter: they are situated at variable distances from each other, often thick-set and touching at their bases. The nodules may be pretty uniform in size, sometimes not larger than hemp-seeds; but in the advanced stages they are often more unequal. On section, the nodular arrangement is found throughout the substance of the organ. The cut surface presents a collection of roundish and darkish patches, varying in size and shape, and separated from each other by intervening firm, whitish, fibrous-looking or cellular tissue: the arrangement has been compared to the vitellarium of the laying hen. The nodules are yellowish, sometimes bright yellow, from bile. Dr. Budd likens the colour to that of impure bees'-wax. This affords a contrast to the whitish intermediate substance. The breadth of the white tissue varies from mere lines to spaces equal to the size of the lobules. The coloured masses are the remaining secreting tissue, lobules or groups of lobules not yet wasted or absorbed. There is thus a remarkable diminution of secreting tissue, and a real or apparent increase of white connective or areolar formation. The wasting of the lobules varies in degree in different parts of the liver.

Microscopic examination shows that great changes have also taken place in the minute structures, and especially in the cell-growth and capillaries of the lobules.

The cell-structure is greatly reduced: not only has the greater part of this entirely disappeared with the lobules, but that left in the remaining lobules is often found in different stages of alteration. A portion of the cells may be found healthy and bile-stained; but the greater number have undergone degeneration, become smaller or shrivelled up, or they may be dotted with or filled with oil. The colour is generally yellow, but some are brownish. The cells of the periphery of the lobules are most wasted or altered.

The circulation of blood in the affected lobules is much interfered with, or entirely arrested, according to the degree of the waste of the secreting structures, owing to the disappearance of, or changes in, the capillary plexuses. The walls of most of those which remain are granular, or contain oil-globules.[1] Fine injections of the portal vein do not penetrate the capillaries of the interior of the diseased lobules. The hepatic veins may be traced into the centres of the lobules,

[1] *Beale's* Archives of Medicine, vol. i. p. 122.

but their capillary ramifications have disappeared : a few branches, however, generally remain, become enlarged, and maintain the connexion of the hepatic with the portal or veins of the periphery of the lobule. Through these changes, the communication between the portal and hepatic veins is nearly destroyed or interrupted, and the blood can no longer easily pass onwards. Even where the vessels in part still remain, the blood-channels are insufficient to continue the circulation fully, though a small quantity of portal blood may still pass onwards. The hepatic artery is said by Frerichs to become enlarged, and to develop new capillaries. Black pigment is sometimes found in its branches.

The white substance varies in quantity and hardness, and is generally supposed to be made up of fibrous tissue; but that this is not always correct appears from a case which was minutely examined by Dr. L. Beale,[1] and which may be supposed to represent, at all events, one form of Cirrhosis. In this instance it was not composed of fibrous tissue resulting from organized exudation, but was made up of numerous vessels, bile-ducts, and tissue consisting of altered and partly disintegrated vessels, cells, and other tissues, which existed in the healthy state of the organ. It was found to be penetrated in every direction by vessels of considerable size, but which, however, stopped short before entering the lobules. The bile-ducts were generally permeable to injections, and were traceable through the white matter. Their walls were granular, or filled with oil-globules.

Other observers describe the white substance in the cirrhosed liver as consisting of newly formed fibrous tissue, originating in marked exudation, which, undergoing contraction, causes compression of the vessels and lessening of vascularity. Frerichs says that, at the circumference of the lobules, fibrillated connective tissue is found, and that in the substance of the lobules the tissue has an amorphous character.[2]

The condition above described is that to which the liver is reduced in extreme states of Cirrhosis—a state of degeneration which it has reached in succession to previous changes only. Pathologists are not wholly agreed as to the processes which always precede atrophy. The same kind of doubt is entertained respecting the early stages of these as of the early stages of the contracted kidney. It has yet to be determined whether Cirrhosis is the result of a primary degenerative process, or of inflammatory exudation or congestion. It is probable that it may originate in either of these processes.

When it commences with inflammation or congestion, the course which leads to atrophy is as follows:—Fibrinous exudation takes place ; this occupies the portal canals, and extends even into their minute ramifications, so that the very lobules may be separated by the exudation. Livers examined in this earlier stage are much enlarged, are firm and tough—sometimes very tough—the external surface,

[1] Beale's Archives, vol. i. pp. 120, 121.
[2] Clinical Treatise on Diseases of Liver, New Sydenham Society's Translation, vol. ii. p. 28.

perhaps, merely uneven with commencing granulations, and the capsule more or less thickened and opaque. On section there is found considerable vascularity, an amorphous albuminous exudation, tailed or spindle-shaped cells and fibro-cellular tissue separating the lobules. In more advanced stages the fibrous tissue is more decidedly developed. Subsequent to the organization of the exudation, contraction follows, with constriction of the vessels lying in the course of the new tissue, impediment to the circulation in the small branches of the portal veins, starvation and wasting of the tissue by them. Dr. Budd takes this view, and describes Cirrhosis as the result of adhesive inflammation of the liver, involving the areolar tissue about the small twigs of the portal veir : by this inflammation serum and coagulable lymph are poured out, causing general swelling of the liver. This exudation subsequently loses its fluid part by absorption, becomes denser, separates the lobules into well-defined masses, and constricts the smaller branches of the portal vein. Wasting of the lobules ensues ; those on the surface, and the islets of coloured tissue seen in sections, are congeries of lobules not yet wasted and absorbed ; the yellow colour depending upon some compression of the ducts, and consequent bile-staining. Dr. Budd thinks that when the exudation and thickening occupy the larger canals or passages, and cause compression of some of the larger branches only of the portal vein, atrophy of the part supplied by the branch is produced, without much alteration of the other portions, shown by drawing in or puckering of the surface.[1]

Those who attribute Cirrhosis to degeneration of the secreting tissue, independently of inflammation, suppose that an unsuitable pabulum passing through the liver, as in the case of the long-continued consumption of alcoholic liquors, produces degeneration of its cell-tissue. This causes a smaller demand for, and a diminished afflux of, portal blood, and wasting and absorption of the lobular structure, leaving masses of white tissue which waste less rapidly than the lobules. That Cirrhosis may be produced in this way seems probable from the description given by Dr. Beale, already mentioned. He shows that, in the case named, the white substance was well provided with permeable vessels, and that the atrophy could not have been in that instance due to the constriction of the vessels merely : nevertheless, as their capillaries were obliterated, portal blood could not circulate in the lobules.

In considering the immediate causes of contraction of the liver, it seems impossible to doubt that in a large number of cases enlargement of the organ precedes the atrophy. Dr. Bright is confident of this. In more than one instance in the post-mortem examination of cases of contracted liver I have myself seen proof of its great previous enlargement in the state of the adhesions connecting it to distant parts. I have seen the left margin of the liver attached to the left origin of the diaphragm by membranous adhesions three or four inches in length, showing that in some former state the edge

[1] Budd, Diseases of Liver, 3rd ed. pp. 144 and 183.
[2] Guy's Hospital Reports, vol. i. p. 612.

had been in contact with the left origin of the muscle, and that during this time exudation upon the surface had taken place, which had been drawn out into thin sheets of adhesions by the slowly retreating liver. It is not unfrequently found that Cirrhosis has been preceded, three, four, or more years before its establishment, by well-marked pain and enlargement of the liver. Malarious enlargements sometimes end in Cirrhosis. Frerichs mentions that in some of these cases the portal capillaries have been found to be blocked up by pigmental deposit, so that they were impermeable to injection. I do not know that there is any good proof that the simple, painless, or nearly painless, enlargements of the liver frequently met with in tropical or malarious climates, unconnected with fatty or albuminous disease, terminate in Cirrhosis. It is probable that they do so sometimes; but it is by no means a necessary consequence. The enlargements from passive congestion, resulting from heart-disease, are, if the patient survives long enough, often succeeded by an atrophied state, which in external appearances resembles Cirrhosis; and if the primary disease destroys life before there is extreme degeneration, the surface of the liver is occupied with small elevations, similar to those of the early stages of Cirrhosis.

It seems probable, then, that Cirrhosis, as before said, may originate in two ways—either as the result of primary degeneration of cell-growth or of inflammatory exudation. In both of these conditions, however, it is possible that cell-degeneration may be the step which originates the wasting of the capillaries of the portal veins, and that even in the inflammatory variety this vital or functional disturbance plays a more important part than mere mechanical compression of the vessels. It is not impossible that in some cases compression of the bile-ducts may produce biliary stagnation and wasting of cells.

The other forms of atrophy of the liver remain to be briefly noticed. Atrophy may be partial, from pressure of tumours from above or below the gland. Depression of the diaphragm, from thoracic disease or from limited peritoneal exudations, may produce local absorption or depression.[1] Diminution of the liver, in common with other organs, attends many emaciating diseases and old age. Tight lacing also causes it. In cases of perihepatitis with much thickening and contraction of the capsule, and consequent general compression, atrophy may follow.

There is a form of chronic atrophy called *red atrophy* by Rokitansky, in contra-distinction to the chronic yellow atrophy. It is distinguished from the latter by the darkish-brown or bluish-red colour of the organ. The liver is gorged with blood, and is of spongy elastic consistency. The outer and inner parts are free from granulations, and the surface of sections is of smooth and homogeneous texture. The organ is atrophied, the thickness preponderating over other dimensions. It is attended with venous plethora of the abdominal viscera. According to Frerichs,[2] the portal vein is enlarged

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[1] Frerichs, vol. i. p. 249.　　　　[2] Ibid. p. 253.

up to its subdivision into capillaries at the periphery of the lobules, at which point the enlargement terminates in club-shaped extremities. The walls of the veins are sometimes normal, but at others there is remarkable thickening of the sheath formed by Glisson's capsule. The hepatic capillaries are in great measure destroyed. They become filled with brown molecules, and contain flakes or granules of black pigment. The lobules are atrophied, and the capillary meshes contain only a few stunted cells. The veins of the stomach and intestines are frequently enlarged, and there may be sub-serous ecchymoses and mechanical congestions of the mucous membrane and of the spleen.[1]

Frerichs points out that the atrophy which follows the *passive congestion* in thoracic disease differs somewhat from true Cirrhosis in its minute anatomy. It has already been mentioned that, in the ordinary granular liver, the cells and other structures waste at the periphery of the lobules; but it is otherwise in passive hyper-æmia. The congestion in this form affects the interlobular branches of the hepatic veins, causing stagnation of blood, distension, and atrophy, which last by degrees extends to the parts supplied by the portal capillaries. In atrophy from passive congestion, the wasting commences at the centre, and in Cirrhosis at the periphery of the lobules. He says that this circumstance gives a different appearance to the sections, the granular state being marked by depression in the centre in the first, and at the circumference in the second case.

Of late years, carefully made observations have shown that constitutional *syphilis*, probably in its tertiary stages, produces contraction of the liver, preceded by interstitial inflammation and exudation. The resulting anatomical changes differ in appearance from those of common Cirrhosis. The surface is not hobnailed, and the atrophy is more localized, causing deep furrows leading to depressed cicatrices, more frequent in the upper than in the under surface of the liver, and dividing the organ into irregular masses. In well-marked cases the liver acquires a lobulated form of unequal divisions, as seen in the 6th figure of Frerichs' second volume, page 166, and in which condition it has been likened to the kidney of the fœtal calf. In the earlier stages the exudation occupies chiefly certain ramifications of the capsule of Glisson, and the subsequent organization and contraction interfere with the nutrition of the parts supplied by the vessels of the tract involved. The exudation ultimately forms hard white spaces of fibrous tissue, or masses connected with the base of the furrows. In these new tissues the gummy syphilitic deposits are sometimes found. The conditions of local atrophy observed by Dr. G. Budd, previously referred to, are probably illustrations of the disease in syphilitic subjects. Perihepatitis with adhesions to the diaphragm is pretty constant. More detailed accounts than can be given here

[1] Frerichs, vol. i. p. 253.

will be found in the descriptions of Frerichs,[1] Wilks,[2] Murchison, Berkeley Hill,[4] and Langereau.[5]

In whatever form the atrophy exists, whether under that of Cirrhosis, red atrophy, or that from passive congestion, the results in the extreme stages are impairment or wasting of the secreting structure, obliteration of the capillaries of the lobules, and obstruction to the current in the portal veins, with distension of its trunk and intestinal capillaries; the ultimate consequences being great reduction of the bile-producing capacity of the liver, portal congestion, imperfect digestion and nutrition, anæmia, and hæmorrhages.

The changes which occur in other organs are in great part due to the obstruction to the portal circulation in the liver. Thrombi sometimes form in the branches or trunk of the portal vein; with or without this there are congestions, ecchymoses, rupture of capillaries or hæmorrhages affecting the stomach and intestines. Perhaps the stomach and small intestines are most liable to hæmorrhage, but they may spring from any part of the canal. Sometimes abrasions and superficial ulcers of the mucous membrane are formed. Congestion of veins of the colon and rectum leads to ulcers, hæmorrhages, symptoms of dysentery, and hæmorrhoids. The disturbance of the circulation of the stomach causes imperfect digestion, difficult absorption, and impaired nutrition of the body. The mechanical impediment to the passage of blood through the liver compels it to find new channels to the heart. These new channels are formed in one or two situations. In one, an anastomosis is formed between the hemorrhoidal and the branches of the internal and the external iliac veins; in another, between the portal veins of the surface of the liver and the veins of the diaphragm and neighbouring parts through the medium of the adhesions connecting the liver with them, and also through some accessory branches of the portal vein which reach the liver through the suspensory ligament, and maintain a connexion with the walls of the abdomen and with the epigastric and internal mammary veins. When the umbilical vein remains pervious, blood flows back from the liver, and forms a remarkable anastomosis about the navel, which communicates with the mammary and other veins. Sappey denies that this really occurs, and thinks that the enlarged vessel in the suspensory ligament, usually taken for the umbilical vein, is the enlarged accessory vein just mentioned. Sappey was the first to point out the nature of this important collateral channel. By the anastomoses above mentioned a considerable quantity of blood may reach the venæ cavæ direct from the intestines. The full establishment of the new circulation is marked by great development of the superficial abdo-

[1] Op. cit. vol. ii. pp. 150—166. Atlas, part ii. plate 4.
[2] Lancet, January 1857, June 1858.
[3] Op. cit. p. 241.
[4] Syphilis and Local Contagious Disorders, pp. 132, 133.
[5] Langereau, Treatise on Syphilis. New Sydenham Society Ed., vol. i. pp. 332—35.

a. The collection may be very considerable. The fluid is
sh, and is rich in albumen.

anæmia depends upon the imperfect nutrition from disturb-
the digestive process, and absorption by the portal veins,
obably also from the disordered function of the liver.

generally supposed that hepatic abscess is seldom or never
n Cirrhosis. I do not recollect meeting with this complica-
ut Dr. Morehead mentions its occurrence in four of his cases.[1]
ere all preceded by dysentery, and may have been examples
mic abscesses following the dysentery.

morbid changes in other organs besides the liver may be
l up as those due to the mechanical effects of the atrophy,
e, as appears from the foregoing, congestion of the portal
of veins, hæmorrhages, erosions or ulcers of the gastric or
al mucous membrane, hæmorrhoids, enlargement of the deep
perficial abdominal veins, and ascites. Those due to bad
on, imperfect nutrition, and sanguification are partly attribu-
o the mechanical derangement, and partly to disturbed liver
n,—as anæmia, purpura, tendency to ecchymoses and hæmor-
from spanæmia, jaundice. The kidney is occasionally con-
sometimes owing to the pressure of the fluid in ascites, and
nes atrophied, probably in consequence of constitutional de-
ent common to it and the hepatic disease.

ometimes happens that Cirrhosis exists with an enlarged liver.
ccurs in cases of fatty or waxy degeneration coinciding with
rrhosis. In such cases the term atrophy applies to the
g tissue, and not to the whole organ.

LOGY.—The immediate causes of atrophy of the liver have
been discussed. The remote causes of those forms not
from tight lacing or wasting disease are, in Europe at all

of residence in tropical and malarious countries; also that aris
from chronic heart-disease. It is supposed that the direct irritat
of alcohol, in its passage from the intestines through the li
causes the interstitial inflammation or enlargement or the
degeneration. It is possible, however, that there is also some co
stitutional vice engendered by spirit-drinking, and that there
more than simple local disease. The wide diffusion of degenerati
processes, as in the heart and kidneys, which frequently co-ex
with Cirrhosis, seems to favour this view. Dr. Handfield Jones thin
that, in some instances, the change is rather of the nature of
degenerative process which causes hypertrophy and condensatic
of the fibrous tissue, than an inflammatory one—a change of
similar kind to that which produces cartilaginoid induration of t
capsule of the spleen, stiffening of the valves of the heart, an
contraction of its orifices.[1]

Age and Sex.—Cirrhosis is generally a disease of middle lif
between the thirtieth and fiftieth year; but cases are sometimes foun
in much younger and much older persons. Bamberger found t
limit of age to be fifteen and sixty-five in his patients. In t
"Pathological Transactions" a case is mentioned in a child of eleve
and Frerichs has seen it in one of ten years of age. Men are m
subject to the disease than women.

SYMPTOMS.—The symptoms of the early stages of Cirrhosis d
pend upon the manner in which the disease commences—up
whether it sets in with active inflammation or not. When it begi
without inflammation, they are very obscure, and the complai
progresses slowly, and for some time without apparent chang
In this form the symptoms are mostly referred to the digesti
organs. There is flatulence, distension, sometimes nausea, and t
other signs of laboured digestion; the appetite generally poor
indifferent. At times the symptoms resemble those of catarrh of t
stomach or intestines. The bowels are irregular, sometimes loose, w
slimy evacuations, and at others constipated. The fæces vary
colour from natural to a light hue; occasionally a firm motion m
consist of patches of light mixed with darker material, attributab
to fluctuation in the amount of hepatic secretion; and sometim
they are covered with a thin layer of mucus. There may be
sense of weight or dull pain in the right hypochondrium, more
less persistent or recurrent; but this may be absent or too slig
to be complained of. These symptoms may continue for month
with more or less severity, in spite of treatment, the patient gettin
slowly weaker, thinner, and more anæmic. Hæmorrhoids frequent
appear: to these succeed distension of the superficial abdomin
veins, ascites, and often hæmorrhage more or less profuse. T
ascites may precede the full enlargement of the veins. The ascit
increases, and anasarca of the lower limbs may come on. T

[1] Jones and Sieveking's Pathological Anatomy, p. 554.

dropsy interferes with the descent of the diaphragm, and this action is often further impeded by flatulence, so that great dyspnœa is caused.

In the early stages the liver may be found larger than natural on percussion; but as the disease progresses the hepatic dulness will diminish, so that, when the atrophy is extreme, a narrow band of dulness is all that remains to indicate the position of the liver. There is sometimes well-marked jaundice, but this is not common in uncomplicated cases; usually, however, there is dinginess or sallow-ness of surface and slight yellowness of conjunctiva. In the beginning the urine may not be much altered; but when there is lessened absorption, owing to congestion of the portal veins, the urine may be much diminished. Dr. Parkes thinks a copious or even natural flow of urine a strong argument against the existence of any material amount of congestion or Cirrhosis.[1] Uric acid and lithates are often precipitated, frequently with reddish or pink colouring-matter. These last, however, are not peculiar to contracted livers. The proportion of urea varies with the character of the digestion. When the liver function is greatly interfered with, as in the later stages, leucine and tyrosine may appear. There is seldom much bile-pigment.

The general aspect of a patient with advanced Cirrhosis is alone almost diagnostic. The thin, pale, sallow, or dingy, pinched coun-tenance, the skin dotted or spotted with petechiæ, the emaciated chest and legs, the swollen, globular abdomen, with the distended veins meandering over its surface, are remarkable and characteristic.

The sufferer may die with hæmorrhages, purpura, dysenteric stools, or general asthenia. In some cases life ends with delirium, sopor, or coma of some days' duration, or with typhoid symptoms, or pneumonia or bronchitis. In many, however, the mind remains clear until the last, the patient dying of exhaustion.

In some patients the first alarming signs are hæmorrhages, as, for instance, severe hæmatemesis repeated or even fatal in the first attack (but this is rare), melæna, or repeated hæmorrhages from some other part of the mucous membrane; sometimes, and perhaps more frequently than is observed, there is slight oozing of blood, which mingles with the contents of the bowels, and may not be recognised except upon microscopic examination. The hæmorrhages may be, and perhaps are, generally due to mechanical congestion; but probably in many instances they are combined with a diseased state of the blood, as they occur also in parts supplied by vessels not subject to portal obstruc-tion, as from the nose and mouth.

In many instances, when the early symptoms are obscure, the first sign which attracts attention is the ascites. This is very characteristic. In typical cases it comes on without previous œdema of the lower extremities, differing in this from the dropsy of heart-disease.

The cases which begin with inflammation or congestion may have decided pain or tenderness in the hepatic region, increased by pressure,

[1] Parkes on the Urine, p. 324.

or by deep inspiration, or by certain positions. The pain depends a great deal on the situation of the inflammation, being most severe when it involves the capsule and the upper surface of the liver. The pain and uneasiness are more marked when the distension of the capsule by the engorged liver is rapid than when it is slow. The pain may last a few weeks, and disappear and return again, or it may exist in a slighter degree throughout the disease, or be more troublesome when the patient is in certain positions—probably when on his left side.

DURATION.—Cirrhosis is a very chronic disease. It often lasts three or four years, or even more, from the commencement of the derangement of the health to the setting in of marked symptoms of obstruction. After this the duration is more definite : it may be a few months or a year.

DIAGNOSIS.—Before the effects of Cirrhosis begin to show themselves the diagnosis is difficult. If with previous symptoms of disturbed digestion there is a history of previous liver enlargement or inflammation, or of spirit-drinking, or of exposure to malaria, a strong suspicion may be entertained that degenerative disease of the liver is going on. When there is considerable emaciation and absence of ascites and flatulence, the lumps or nodules may sometimes be felt through the skin. In some instances, and especially among hospital patients, there is less difficulty in coming to a conclusion, as the patient does not seek medical advice until the characteristic symptoms are well developed. In the early stages of the insidious forms, the disease often passes for aggravated permanent dyspepsia. It is well in all these persistent cases to examine carefully into the state of the liver itself, and of its secretions, the tendency to piles, hæmorrhage, melæna, slimy motions, or microscopic intestinal hæmorrhages. In ordinary dyspepsia the disease may last for years without much emaciation, but this is more marked in indigestion accompanying hepatic disease. The size of the liver should always be carefully inquired into, but it is only in the later stages that much additional information is derived therefrom. Of course, when a case is under observation from beginning to end, it may be possible to trace its progress from period to period; but this opportunity is not often found.

In advanced cases it is not probable that much difficulty will occur. The ascites must be distinguished from ovarian dropsy, hydatid cysts, effusion from peritonitis, and the ascites of general dropsy. From ovarian dropsy it differs in the position of dulness and the history of growth; from peritoneal effusion, by its coming on without previous symptoms of acute or chronic peritonitis; from general dropsy, by the abdomen becoming swollen first and the lower extremities subsequently, the absence of general puffiness or œdema of the surface, and of heart, lung, spleen, or kidney disease, though these may occasionally co-exist. The condition of the superficial veins will help

the diagnosis. Extreme collection of fluid in the peritoneal cavity from any cause may cause prominence of these veins: but in contracted liver the enlarged veins generally precede the ascites ; in general anasarca the fulness of the veins comes on as a consequence of the pressure of the fluid on the inferior cava. The portal origin of the ascites being ascertained, there may be difficulty in discriminating between obstruction or obliteration of the trunk of the portal vein by thrombosis or the pressure of tumours or growths, or by the obstruction in the liver itself. The diagnosis may be assisted by the knowledge of the size of the liver, by the previous history of disturbed liver function, by the presence or absence of tumours pressing upon the vein. In thrombosis the obstruction is sudden, and the enlarged lower abdominal veins will be more rapidly developed than in Cirrhosis; the fluid collects again after paracentesis, even more rapidly than in Cirrhosis.

The size of the liver can sometimes be ascertained by repeated examinations only, the bowels being cleared of fæcal accumulations and flatulence by aperient medicines or stimulating enemata. Its condition should be carefully investigated after paracentesis. It would not be possible to diagnose between the various forms of atrophy, unless, indeed (as may occasionally be done), the nodules could be felt.

PROGNOSIS.—This is decidedly bad. Fatal results may not be immediate, but sooner or later they must be feared. When the disease is sufficiently developed to make diagnosis certain, it is, as far as is positively known, always progressive, though its rate may vary much. It is probable that in patients in whom very free collateral circulation is established the progress may be slower, as cases are on record in which ascites has disappeared when the various venous anastomoses have been largely formed. It is possible that in such cases of free restoration of circulation by means of collateral channels, if the uninjured portion of the liver be sufficient for the purposes of life, and the atrophy arrested, that existence may be maintained ; but there does not appear to be good evidence of such cases and results having occurred. I suspect that the downward progress of the disease will be slower also in those instances in which easy, slimy, mucous motions relieve the congested mucous membrane.

TREATMENT.—It is only in the early stage, in that of enlargement of the liver, that we can hope to arrest the disease by treatment. Remedies which relieve congestion and inflammation should be used. In the acute stages, leeches, blisters, and saline aperients. In the chronic stages of enlargement, iodide of potassium, muriate of ammonia, nitro-muriatic acid, internally ; or, externally, fomentations, pediluvia, or baths with nitro-muriatic acid should be tried. In syphilitic forms, mercury or iodide of potassium may be used, according to the patient's condition. Change from tropical or malarious countries to temperate ones, if chronic enlargement continues, should be resorted to. It is not that these chronic enlargements necessarily

end in Cirrhosis; but it is well not to run the risk in any case. As Cirrhosis so often springs from spirit-drinking, it is imperative to prohibit its use in any form, except under rare circumstances, and to prescribe a bland, nutritious, and unirritating and easily digestible diet. Hot spices are to be avoided. They are generally objected to in Europe; but, it must be remembered that the natives of India consume them continually and continuously, without suffering particularly from Cirrhosis.

When extensive degeneration of the liver has occurred, health cannot be restored, and it becomes a question how far the remaining tissue can be kept in its existing state, and how the symptoms may be palliated. We do not know how much healthy liver-tissue is necessary for existence; but it is probable that a large destruction of it is compatible with the maintenance of life. We can only hope to effect this object by supporting the general health, which may be summed up as consisting in the use of tonics, such as iron, quinine, strychnine, and pepsine, to assist the digestion. The skin should be kept warm, and warm baths occasionally used. Rest from business and care is necessary. It would be vain to enter into a description of the remedies for the various symptoms of acidity, flatulence, heartburn, hæmorrhages, scorbutic, dysenteric, or typhoid conditions, or brain-disturbances. They are such as are usually applied in such cases. The ascites rarely yields for any time, though it may be lessened or be kept temporarily under by diuretics or purgatives. The most useful diuretics are squill, digitalis, and decoction of broom tops. The latter sometimes acts powerfully, when others fail. The combination of blue pill, digitalis, and squill is a favourite remedy with some practitioners. The mercury should never be given when the patient is in any degree in a cachectic state. Purgatives are not so valuable in this as in ordinary dropsy. I have found that, when there is permanent portal congestion, they tend to produce irritation of the mucous membrane, scanty, mucous, or dysenteric stools. Still they should be tried cautiously. The compound-jalap powder in drachm doses may be given, and stopped if it produces straining or scanty evacuations: also elaterium in small doses.

When the collection of fluid is large, and the breathing difficult, tapping may be required, and is, indeed, the only remaining means that we possess for giving relief. It should not be too quickly resorted to, because, unfortunately, the fluid collects again very rapidly, and the operation requires speedy repetition, often in a week or ten days. Probably, the fluid exudes all the more rapidly, owing to pressure being lessened by the evacuation. It is better, however, to tap than to allow the patient to go on with great dyspnœa or suffering. This is of itself a very exhausting process,—perhaps equally so as the loss of albumen caused by the rapid secretion. When the strength is good, an ordinary but small-sized trocar should be used; but I have for years, in cases of ascites of any kind, with great debility, employed for evacuating the fluid the small needle and canula used

for exploring purposes, and which is not much larger than a worsted-needle. The patient lies on his side in bed, and the fluid escapes slowly, and without causing any exhaustion; gentle pressure being maintained. A piece of india-rubber tubing, slipped over the shield of the canula, serves to conduct the fluid into a basin, which may thus be collected without fatigue to the patient, and without wetting the bed.

ACUTE OR YELLOW ATROPHY OF THE LIVER.

By EDWARD GOODEVE, M.B.

DESCRIPTION.—In this fatal disease there is rapid diminution and change of structure of the liver, with jaundice. It is very rare, and has already been alluded to, in the article on Jaundice, as one of the malignant forms of that disease. It has been accurately known for a few years only, having been first described by Rokitansky, in 1845.

SYMPTOMS.—These are much the same as those of malignant jaundice, and will therefore need a brief notice only in this place. The jaundice may begin suddenly, and run a rapid downward course, or there may be at the outset a few days of preliminary disturbances. About half the cases begin in one way, and half in the other.

In those with preliminary stages the earlier symptoms may be mild, and give no indication of impending danger. The patient may, indeed, not seem to be really ill. The symptoms are debility, weariness, aching of limbs, disturbances of the digestive organs, nausea, vomiting, flatulence, pain and uneasiness in the epigastric or hepatic regions, constipation or diarrhœa, slight feverishness, or quickening of the pulse. After a few days' duration, these may be followed by jaundice, which is generally not so deep as in obstructive jaundice, and most marked in the upper parts of the body. In this state the alvine evacuations are not devoid of bile. So far the disease may, in severity, differ little from catarrhal or simple jaundice. While in this state, and apparently with but little warning, severe disturbances of the nervous system suddenly appear: these may be agitation, restlessness, jactitation, delirium, sopor, profound coma or convulsions, with involuntary stools and urine. The evacuations, as above stated, generally contain bile, but they may become clay-coloured or white as the disease advances. There come on deep alterations of the blood, vomiting of dirty brownish fluid, or of grumous or coffee-grounds matter, hæmorrhages from the intestinal canal and vagina, extravasations of blood, and petechiæ. The feverishness generally subsides, and the pulse falls, when the grave symptoms appear. Pressure on the liver during the insensibility often causes contraction of the muscles of the face and signs of shrinking from it. The urine is generally diminished, but not suppressed, is of dark colour, contains bile-pigment, tyrosine, leucine, and less than

the normal amount of urea and uric acid. Dr. Harley says that the
acids may be found in the urine in this variety of jaundice; so that
the jaundice is partly due to suppression and partly to absorption.
Under the above complication of disease of the blood and brain the
patient generally sinks. Towards the last, the pulse loses in strength
and gains in frequency, and is liable to great fluctuation in number.

When the disease has made progress, the liver becomes much
diminished in size, which is indicated by the progressive lessening of
the hepatic dulness if there be time for daily examination. On per-
cussion in the later stages, it is found to be reduced to a narrow band
about an inch in breadth, in the hepatic region.

MORBID ANATOMY.—It has been already mentioned, in the paper
on Jaundice, that the liver is much diminished in this form. It
has lost half or a third of its weight and size. The thickness is greatly
reduced, often not exceeding an inch at its greatest. The liver looks
as if flattened out; its edge is thin. The capsule is shrivelled or
puckered; not thickened or infiltrated with exudation, but marked
with ecchymoses and subperitoneal extravasations. The tissue is
generally soft and flabby, and easily broken down, or even pulpy.
Portions may be congested, and less wasted than the rest. On section,
the colour is orange-red or yellow or rhubarb-colour, sometimes
greenish yellow; some parts are pale, others dark and congested. The
divisions of the acini or lobules have disappeared; the cell-structure is
broken up, and in most parts destroyed; those cells which remain
contain much oil, and are sometimes granular. In cases cut short
before the full changes are completed, as in the case of death by
hæmorrhage in an early stage, part of the cells may be but little
altered, while the others have disappeared and are replaced by oil or
granular detritus. Crystals of tyrosine and leucine are met with in
the liver-substance. Frerichs speaks of a dirty greyish exudation
being found in some parts of the organ in the earlier stages, but which
has disappeared in the later ones : it is found between and separating
the lobules, which are surrounded by congested vessels. Injections by
the hepatic or portal veins stop short of the capillaries, and between
the ramifications of these lie the disintegrated tissues and extravasated
injection-matter.[1] The effect of the morbid process which the liver
goes through is found in extreme cases to be the complete destruction
of its secreting structure, disintegration and partial absorption of its
component tissues, with the production of oil or fat.

The gall-ducts and gall-bladder are generally empty, or the latter
may contain a little unhealthy bile or a little grey mucus. The
passages are not obstructed.

The blood is generally altered in quality, coagulating imperfectly,
and containing, especially in the hepatic veins, and sometimes in the
cavities of the heart, tyrosine and leucine.

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[1] Frerichs, New Sydenham Society's Translation, vol. i. p. 225.

The spleen is pretty constantly enlarged, as are the kidneys; the epithelium of the latter being bile-stained and fatty.

Extravasations of blood, ecchymoses, and changes in the other organs are such as are met with in other states of malignant jaundice.

In a few instances the liver has been found in a state resembling cirrhosis. In these cases the tuberculated condition has probably not been caused by the acute disease, but this last has come on in a patient who was the subject of cirrhosis.

DURATION.—The duration of the stages is uncertain. The milder symptoms may last from two or three days to three or four weeks. The jaundice may not come on for some days after the appearance of the preliminary signs, and may itself precede the severe symptoms for some days. Sometimes, however, the jaundice sets in early, and with it grave cerebral symptoms rapidly come on.

In those cases in which there may be said to be no preliminary symptoms, the jaundice and coma may be the first symptoms observed, and the patient may continue in this state and die in the course of two or three days, or even less. The whole disease may be said to last from one or two days, in very rapid, to three or four weeks in the extremely slow cases.

ETIOLOGY.—The causes of acute yellow atrophy are involved in great obscurity. The disease has been known to attack several persons in the same locality, and sometimes to have put on a sort of epidemic character, suggesting the idea of the operation of blood-poisoning from some local or general external cause. In many cases it has followed mental emotions, such as fright, grief, despondency, and also venereal excesses and debauchery of various kinds.

There are two distinct points to be considered in the etiology of the disease: first, the cause of the liver-disease; and, secondly, the cause of the malignant symptoms. Dr. Budd thinks that the liver-disease is caused by some noxious matter absorbed from the intestinal canal, either swallowed with the food or produced within the body through faulty digestion or assimilation.[1] Frerichs saw it in a case resembling abdominal typhus in its earlier symptoms. Dr. Murchison thinks it probable that the process leading to liver-atrophy is part of a general constitutional disturbance due to some blood-poisoning, as in typhus or other blood-diseases, and in the same way that fatty change in the liver is only one of a series of changes following phosphorus-poisoning.[2] Although in the present state of our knowledge it is impossible to pronounce with certainty on this point, there is much probability in it; but it is also quite possible that the liver-disease may be due to blood-poisoning, and yet be the only local change that may be called primary, while the alterations in the other organs may be secondary.

The question of the cause of the malignant symptoms has been

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[1] 2nd Ed. op. cit.   [2] Lectures on Diseases of the Liver, p. 346.

treated in the section on Jaundice. They are probably due to the blood-changes associated with renal derangement.

The intimate nature of the morbid process leading to the diminution of the liver is a subject of doubt. Frerichs thinks that the liver-disease is essentially a diffused or parenchymatous inflammation, with scanty exudation and fatty degeneration of the cells and other tissues of the lobules, leading to disintegration and absorption of the degenerated structures. The earlier steps of the process, however, have not been proved. Frerichs speaks of a greyish exudation in certain parts of the liver examined in the earlier stages, and which disappear in the later ones. Professor Bamberger and Dr. Murchison attribute the atrophy to diffused inflammation and rapid fatty degeneration of the tissue. If Frerichs is correct, the atrophy should be preceded by some enlargement during the exudation stage. This condition has not been observed during life; but, as he states truly enough, attention has not been sufficiently directed to the early state of the liver, to enable that point to be determined. Wedl speaks of the disease as a diffused hepatitis, in which the parenchyma of the organs is softened in consequence of an albuminous exudation.

Age and Sex.—The young are more frequently attacked than the old or middle-aged. Frerichs considers the majority of attacks occur between the ages of twenty and thirty.[1] Women are more subject than men; and pregnant females suffer more than others of their sex.

DIAGNOSIS.—This is founded upon the general signs of jaundice, as mentioned in that article, and upon the size of the liver and rapid decrease under examinations, as ascertained by percussion. It may be mentioned, however, that it is difficult, even in a spare person, to ascertain the real size of the liver, and that the investigation requires great care. The occurrence of tyrosine and leucine greatly assists the diagnosis, and should always be sought for when possible to collect the urine.

PROGNOSIS.—This is bad. A very small number of cases of malignant jaundice recover, and it is doubtful if those which have done so were instances of yellow atrophy. In the earlier stages, the signs are not sufficiently established to enable an opinion as to the real nature of the disease to be formed with confidence.

TREATMENT.—Little more can be said on this point than has already been done under the head of Malignant Jaundice, and the reader is therefore referred to that article. It is obvious that, if any success is to be expected, it must be in the early stages. When almost universal or extensive alteration and destruction of the secreting structure of the liver have taken place, no remedies can be of avail.

[1] Lectures on Diseases of the Liver, vol. i. p. 234.

FATTY LIVER.

By J. WARBURTON BEGBIE, M.D., F.R.C.P.E.

GENERAL HISTORY.—Fatty liver, or fatty degeneration of the liver, is one of the recognised forms of painless enlargement of the liver, the morbid change being due to the abnormal deposition of oil or fat in the organ. To the existence of this particular disease attention was first prominently directed by the French physicians, and more especially by M. Louis, who had found it to be a not uncommon accompaniment of phthisis.

By French writers it has been styled " La Transformation graisseuse du Foie," " État gras du Foie." By the Germans it is known under the names of " Die Fettleber," " Hepar adiposum."

There exist very considerable difficulties in the way of offering an accurate clinical history of fatty liver, owing to the frequent absence of those features which indicate during life the precise nature of the hepatic enlargement, in particular instances. It is on this account that Louis is found expressing himself as follows :[1]—" Toutefois, si j'admets que le passage du foie à l'état gras peut être aigu ou chronique, c'est seulement à cause de la dépendance qui existe entre cette lésion et la phthisie ; car nous manquons de signes capables de la faire reconnaître à une époque quelconque de sa durée. En vain j'ai été au-devant des symptômes qui pourraient lui appartenir, je n'en ai recueilli aucun : les malades n'éprouvaient pas de douleurs dans l'hypochondre droit ; la pression sur le foie, quand il dépassait le bord des côtes, n'en produisait pas davantage ; et si elle était douloureuse à l'épigastre, dans les cas où le foie l'occupait, on en trouvait la raison dans l'état de la membrane muqueuse de l'estomac. Une seule fois j'ai vu la couleur de la peau altérée." Again, a little further on, in the same connexion, Louis remarks :—" Dans cette absence de symptômes propres, une seule circonstance peut faire soupçonner l'état pathologique du foie ; je veux parler de l'augmentation de son volume, puisqu'elle existe presque toujours dans le cas dont il s'agit."

The first edition of Louis' celebrated work was published in 1825, and the second in 1843, in both of which the passage now quoted,

[1] Recherches anatomiques, pathologiques et thérapeutiques sur la Phthisie. Deuxième édition, p. 119.

with a slight variation in the latter, occurs. Nine years subsequently to
the appearance of the earlier edition, Dr. Addison, in the course of his
interesting " Observations on Fatty Degeneration of the Liver," took
occasion to quote some of these observations, prefacing them with the
remark :[1] " We find the utter barrenness of diagnosis upon this matter
well illustrated in the candid avowal of the accomplished and ex-
perienced M. Louis." Dr. Addison further observed : " This want of
information is, probably, in a great measure attributable to our in-
ability to recognise the disease during the life of the patient; for as
we then entertained no suspicion of its existence, we fail to make the
minute and connected observation of facts so necessary to the eluci-
dation of every internal affection." And much more recently, Frerichs,[2]
in his valuable work, in directing attention to the subject of fatty
liver, refers to the observations of Louis already quoted, and in doing
so admits their appositeness.

In endeavouring to determine the precise nature and pathological
importance of fatty liver, it is necessary to hold in remembrance that
in the healthy liver of man there is always present some amount of
uncombined oil or fat. The amount of fat in the liver of healthy
adults is probably equal to three or four per cent. of the whole liver-
substance. This fat is contained in the secreting cells of the liver;
and these cells, at certain times and under certain conditions, are
constantly filled with fat. In the fœtus, usually a large quantity of
fine oil-globules is to be found in the cells of the liver; these are
scattered through the interior of the cells, and do not run together so
as to form large drops, as is frequently noticed in the more advanced
periods of life. Frerichs observes that he has frequently examined
the fœtal liver in man and the lower animals, and repeatedly,
although by no means invariably, noticed oil-globules " Fettetröpfchen "
in the cells. From this circumstance, namely, the varying amount of
fat in the fœtal liver, Frerichs concludes that the fatty contents rise
and fall in relation to certain definite periods of development; and
that these are also possibly influenced by pathological conditions.
After birth, and as life advances, the variation in the amount of the
fatty contents of the liver is determined by many different causes.
Of these diet is one of the most important.[3]

[1] Guy's Hospital Reports, first series, vol. i. page 479 : 1836. See also a Col-
lection of the published Writings of the late Thomas Addison, M.D., page 99 (New
Sydenham Society, 1868.)

[2] Klinik der Leberkrankheiten. Erster Band, Seite 285. 1858. " Vergebens,"
observes Frerichs, " war man indess bemüht, nach klinischen Beobachtungen ein klar
gezeichnetes Krankheitsbild für diese anatomische Läsion zu entwerfen : so vielfach
man derselben bei Leichenöffnungen begegnete, so wenig zuverlässige Anhaltspunkte
gewann man für die Diagnose während des Lebens ; eine genügende Symptomatalogie der
Fettleber liess sich nicht construiren. Est gilt in mancher Beziehung noch heute, was
Louis vor Jahren in seinen Recherch. sur la Phthisie hierüber aussprach."

[3] " Anciently," remarks Dr. Thomas Willis, " there was an art with the *Romans* so to
feed a goose that the liver, prodigiously increased, might weigh more than the whole
body."—" Of other Hepatick Remedies,' section 2, chapter ii., of *Pharmaceutice
Rationalis ;* or, " Au Exercitation of the Operations of Medicines in Humane Bodies."
(London, 1679.) This art, as is well known, is still practised. Baron Larrey gives the

The well-known experiments of Magendie demonstrated the fact that, by feeding dogs on butter, the liver became very fatty. In one of Magendie's experiments a dog was kept exclusively on fresh butter, which it continued to eat, although not with regularity, for sixty-eight days. The animal then perished from inanition. Nevertheless, it was remarkably fat. While the experiment lasted, the dog had a strong odour of butyric acid, its hair was greasy, and its skin covered with a layer of fat. On dissection, the whole of the organs and tissues of the body were found infiltrated with fat. The liver was fatty, and yielded on analysis a large quantity of stearine, but little or no oleine: it had acted as a kind of filter for the butter. The general results of the experiments of a like nature undertaken by Magendie were, that the animals became very fat, but their muscles much wasted; in some of them the cornea sloughed, and the animals ultimately perished from inanition. In all of them, the liver was found to be fatty. In connexion with the fatty condition of the liver, Magendie noticed that the skin of the dogs assumed an oily character, and that volatile fatty acids were secreted by the sebaceous glands. This observation of Magendie is of much interest in relation to the description which Dr. Addison gave of the peculiar appearance presented by the integuments in those persons who were suffering from fatty degeneration of the liver in a marked degree. This appearance, when well marked, Dr. Addison regarded as "indicative, if not pathognomonic, of fatty degeneration of the liver."[1] To this condition of the integuments, as described by Dr. Addison, attention will be directed, when treating of the symptomatology of fatty liver. It is not merely food rich in fat which determines such a condition of the liver as Magendie described, but other kinds of food, when taken in too large amount. In such circumstances, however, it has been observed that the deposition of fat in the liver does not occur until the cellular tissue and other organs and tissues of the body have become loaded with fat, and the serum of the blood has assumed a milky appearance. There is such a thing as a fatty condition of the liver stopping short of a truly morbid state; for the

following account of it :—" To procure the large livers of geese for the making of patties, fatted birds are confined in close cages, and then exposed to a graduated heat, being kept at the same time entirely without food—even without water. They become feverish; the fat undergoes a kind of fusion, and the liver grows enormously large. The liver is considered to be in the desired state when the animal is *extremely wasted*, and the fever increases." Upon this account of the late Baron Dr. Budd comments as follows:— " Baron Larrey was a native of the South of France, and the account cited was probably derived from personal observation; but at present, as far as I can learn, there is in the fattening of ducks and geese in France only one method employed, which consists in keeping the birds in a dark place, with little space to move in, and in cramming them with a paste of maize or some other farinaceous food, allowing them water to drink at will. Under this treatment there is at first a general increase of fat in the body; but when the fat stored up throughout the body has reached a certain amount, the further increment of fat appears to be deposited chiefly in the liver, which soon passes into the fatty state that is so much prized. The colour of the fat in any particular bird varies with the colour of the maize on which it was fattened."—Diseases of the Liver, p 303.
[1] Dr. Addison's Works, p. 101.

er is from time to time found in persons who have died suddenly,
e in unimpaired health. It is in these instances a temporary or
ing phenomenon. Frerichs mentions that he has found the liver
fatty ("sehr fettreich") in a railway functionary who was killed at
employment, also in a mason who had perished by a sudden and
ent fall; and the same writer states that he has several times
nd a similar condition in those who have died after a few days'
ess, in the eruptive stage of the acute exanthemata, scarlet fever,
l measles.[1] We look for the occurrence of fatty liver in persons
o lead indolent lives, and are at the same time gross feeders, more
rticularly if their consumption of fatty articles of food is con-
lerable. In individuals of this class the deposit of fat in the liver
almost invariably associated with an excess of subcutaneous fat, as
ll as its increase about the kidneys, in the omentum, and those
her places where its presence is common. In intimate relation
ith the quality and quantity of food, there are other circumstances
quiring consideration, namely, climate and peculiarities of consti-
tion. Fat cannot be taken by certain persons without producing more
less disorder of the digestive and assimilating functions; others are
le to take fatty articles of food without any apparent suffering or
rangement being caused, but they remain lean—the fat is either not
gested or not assimilated by them; a third class not only take such
ticles of food, but, by their use, grow fat. Before passing from the
nsideration of the influence on the development of fat in the
nomy, and specially its deposition in the liver, produced by diet, it
necessary to state specifically, that persons who drink immoderately
f alcoholic liquors, and at the same time take little exercise, are apt
become affected by fatty liver. Dr. Budd[2] mentions "porter and
her heavy malt liquors" as producing this effect. Dr. Murchison[3]
alizes "ardent spirits," and states regarding two fatal cases of
lirium tremens in which an autopsy was made by him in the
ddlesex Hospital, some years ago, that in both there was consider-
le fatty enlargement of the liver. Dr. Begbie[4] has referred to the
uence exerted by "the continued daily use, for a few weeks at a
e, of a mixture of spirits and water, with sugar," in producing
er derangement, and with it a manifest increase of fat, which sub-
ed on the disuse of the stimulating drink. Frerichs, in alluding to
frequency of fatty liver in relation to different diseases, after
gning the first place to tubercular disease of the lungs, remarks,
xt in order comes the drinker's dyscrasia ("Säuferdyscrasie"). Of
teen individuals who died of delirium tremens, the liver was very
y in six ("eine sehr fettreiche Leber"), in three the organ contained
e fat, and in two there was no fat: finally, two died of cirrhosis of
liver.

e turn now to consider a little more fully the relation of fatty

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[1] Note at foot of p. 291 of Vol. i.   [2] Diseases of the Liver, p. 295.
[3] Clinical Lectures on Diseases of the Liver, p. 48.
[4] Contributions to Practical Medicine, p. 214.

liver to phthisis, and particularly to pulmonary tuberculosis, a disease uniformly attended by much wasting and emaciation, and by the disappearance, in particular, of all subcutaneous fat.

The frequency of the occurrence of fatty liver in phthisis was first noticed by Louis.[1] It existed in one-third of the subjects—in forty out of one hundred and twenty. The same writer remarks, " La transformation graisseuse du foie existe presque uniquement chez les individus atteints de phthisie ; en sorte qu'on peut, jusqu'à un certain point, la considérer comme une dépendance de cette affection." The ultimate relationship of fatty liver and tubercular disease of the lungs is further evidenced by the statement of Louis, that, of two hundred and thirty persons who died of acute and chronic diseases, other than phthisis, in nine only was the liver found to be fatty, and of these nine there were seven in which a certain number of tubercles existed in the lungs. The fact, brought to light by the distinguished French physician, has been confirmed by many subsequent observers. Dr. Budd remarks, " Fatty degeneration of the liver, in a high degree, is not only frequent in phthisis, but—setting aside the persons in whom the liver is loaded with fat in common with the areolar tissue and other parts of the body in which fat is liable to be deposited—is almost peculiar to this disease."[2] Frerichs observes, " Among the pathological conditions which influence the origin of fatty liver, tuberculosis of the lungs occupies the first place."[3] Of one hundred and seventeen cases of this kind, in seventeen cases the infiltration was extreme (" höchsten Grades "), and in sixty-two the liver-cells were rich in fat. Dr. Murchison, in fifty-two autopsies of persons dying from tubercle, found the liver fatty in twenty. And the same writer speaks of the " great frequency of fatty enlargement of the liver in persons suffering from pulmonary consumption."[4]

There are a few further particulars of interest to note in reference to this subject. Fatty degeneration of the liver in phthisis occurs irrespective of age, and appears to be of equal frequency in the acute and chronic cases of the malady. Not so, however, in respect to sex. " Le sexe est encore," Louis observes, " une des causes qui favorisent la transformation graisseuse du foie." Of forty-nine instances of fatty liver in tubercular subjects, only two were males. " Das weibliche Geschlecht ist häufiger mit dieser Veränderung des Leberparenchyms behaftet als das männliche "[5] is the statement of Frerichs. And he gives the proportion of males as one to 3·5, while that of females is one to 2·2. So also Dr. Murchison remarks, " In consumptive females it (fatty liver) is much more common than in males."[6]

It is of importance to notice the remarkable contrast offered by the relationship of fatty liver to other diseases of the lungs than the

[1] " La transformation *graisseuse* du foie était la lésion la plus fréquente et la plus remarquable de ce viscère."—Op. cit. p. 116.
[2] Op. cit. p. 298.　　　　　[3] Op. cit. p. 309.
[4] Op. cit. p. 48.　　　　　[5] Op. cit. p. 309.　　　　　[6] Op. cit. p. 48.

tubercular. Neither in pneumonia, nor in pleurisy, nor in emphysema is a fatty condition of the liver observed with any frequency. Formerly, indeed, it had been supposed that the interference to the due performance of the respiratory function, owing to the disease of the lungs existing in phthisis, was the cause of the accumulation of fatty matter in the liver in cases of that disease.[1] This view, however, was rendered very improbable, when it was discovered that, in diseases of the heart which produced great interference with the respiration, in asthma, and in catarrhal disorders, severe and lasting, the liver did not become fatty. The fact now established is, that the fatty condition of the liver is intimately connected, not with pulmonary disease, out with a particular form of pulmonary disease; that the fatty liver is, in the words of Rokitansky, "an essential constituent or pathognomonic combination of the tubercular dyscrasia, inasmuch as it allies itself with tubercular affections of every kind, with tubercle of the intestinal mucous membrane, of the bronchial glands, the serous membranes, the bones, &c."[2]

There are other diseases, attended by much wasting, with which fatty liver is associated. Cancer is one of them; so is the simple ulcer of the stomach; and chronic dysentery is a third. As to the first mentioned, it has been frequently observed that in persons suffering from cancer, although much emaciated, fat has accumulated in the immediate neighbourhood of the cancerous development. Cruveilhier noticed this; and Dr. Budd has stated, that "the most fatty liver that has fallen under my own observation for several years was that of a man who died in King's College Hospital, in April 1844, at the age of thirty-six, of extensive cancerous ulceration of the groins."[3] Dr. Murchison refers to cases of simple ulcer of the stomach connected with fatty liver, as recorded by Mr. R. Robinson (Pathological Transactions, vol. iv. p. 133) and by Mr. H. Thompson (in the same work, vol. vi. p. 186). With chronic dysentery, fatty liver has been found associated by Dr. Bright (Hospital Reports, vol. i. p. 117), by Mr. Busk, and Dr. Budd. To one other, and that a very interesting association of the enlarged and fatty liver with a particular form of disease, reference must be made. "Of the frequent co-existence," writes Dr. West, "of the enlarged and fatty liver with spasm of the glottis there can be no doubt."[4] Of this association Mr. Hood offered a mechanical explanation,—the enlarged liver, in his opinion, leading to the trouble of respiration by the impediment offered to the descent

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[1] The following passage, for example, occurs in the treatise of Andral:—"Presque tous les cas de dégénération graisseuse du foie s'observent chez des phthisiques, c'est-à-dire, chez des individus dont le sang n'est plus convenablement élaboré, et dont l'exhalation pulmonaire ne peut plus s'accomplir comme dans l'état normal; serait-ce parce que chez les phthisiques une suffisante quantité d'hydrogène cesse d'être expulsée par la muqueuse bronchique sous forme de vapeur aqueuse, que chez eux ce principe vient à se séparer en excès de la masse du sang au sein du parenchyme hépatique? De là, formation de matière grasse dans le foie."—*Cours de Pathologie Interne,* tome deuxième, p. 369.

[2] A Manual of Pathological Anatomy, vol. ii. p. 120, Sydenham Society's edition.

[3] Op. cit. p. 299. [4] Lectures on the Diseases of Infancy and Childhood, p. 184.

of the diaphragm. Dr. West, however, after referring to some observations of Dr. Rolleston, which connected fatty liver with the hydrocephaloid disease of children, points out the much more probable dependence of the nervous disorder, in both cases, on the imperfect depuration of the blood in the disordered liver.

APPEARANCE AND CHARACTERS OF FATTY LIVER.—The organ is enlarged,[1] the increase in size taking place for the most part in the lateral direction. The form of the liver is generally preserved to a considerable extent; its surface is smooth, its edges flattened and swollen, the lower margin of the organ being sometimes more distinctly rounded. An enlarged fatty liver may be distinguished from a waxy enlargement of the liver, by being less resistant to pressure and of softer consistence than the latter. The fatty liver is flabby, and on that account is, during life, capable of being pushed aside by the fingers, and it readily ruptures on pressure directly applied. The peritoneal covering is smooth, shining, and tense. In colour, the fatty liver is uniformly yellowish red or light yellow. Rokitansky compares the colour to that of autumnal foliage. The paleness of the liver is associated with a diminished quantity of blood, or anæmia. The existence of fat or oil in the liver is easily proved by the deposit which is left on the dry warm blade of a knife by which it has been cut, or by subjecting the organ to an elevated temperature. In reference to the quantity of fat which is deposited in the advanced stages of the fatty degeneration of the liver, Frerichs remarks that it may be very considerable (" sehr beträchtlich "); and the same author mentions one case in which the amount of fat was equal to nearly four times that of the other portions of the organ.

The essential morbid condition in fatty liver is described by Rokitansky as consisting " in a deposition of free adipose tissue to such an extent as not only to replace the true glandular structure, but to penetrate the entire parenchyma, to the exclusion of the vascular tissue."[2] But while this condition in its exquisite or notable degrees is readily recognised, there are minor varieties of the same, in which the size, form, colour, and consistence of the liver are altered in such a way as to be by no means readily distinguished from certain other morbid changes to which the organ is subject. Accordingly, recourse in such instances must be had to a careful microscopical examination.[3] In fatty liver the deposit of fat takes place in the secreting cells: to these, indeed, it is always limited.[4] In the preparation of a portion

[1] It is not always so : the liver may be diminished in size and weight. Frerichs observes, " Neben den voluminöseren Fettlebern kommen atrophische vor, welche an Umfang wie an Gewicht weit unter der Norm stehen, obgleich ihr Fettgehalt ein sehr bedeutender ist. Solche atrophische Formen gehören keineswegs zu den seltenen."— P. 304. [2] Op. cit. p. 120.

[3] Frerichs remarks, " Volle Gewissheit des Urtheils giebt hier nur das Mikroskop." Op. cit. p. 302.

[4] Such, at least, is the ordinarily received opinion. Frerichs expressly states that he has never discovered deposits of fat otherwise than in the cells. " Die Fettablagerung in

of fatty liver for examination under the microscope, it is of importance to hold in view that the hepatic cells are apt to be injured; thus the fat-globules escape, and appear to lie external to the cells. At first the fat is deposited in the form of fine little drops in the interior of the cells, and ordinarily in proximity to the nucleus, but sometimes in other parts of the cell. These fat-drops increase in number and size, and approach one another; the granular and brown molecules diminish, and the nucleus becomes invisible. Lereboullet and Frerichs differ as to the behaviour of the nucleus: the former believes that he has ascertained the nucleus to perish when the deposit of fat has reached an advanced stage, while the latter affirms that, on the removal of the fat by the addition of oil of turpentine, the nucleus in most instances becomes apparent, and only disappears when the accumulation of fat is excessive, and not always even then. Ultimately the little drops of fat coalesce, forming two, three, or four larger drops; and these again often run together, so as to form one very large drop, almost filling the cavity of the cell.[1] The cells enlarge through the accumulation of the fat, and their form becomes altered; they cease to be angular, and are observed to be more round. As the fat increases, the other contents of the cells diminish and disappear—the fine granular matter and the albuminous substance, and, above all, the brown or yellow granules and globules, which are the product of the secreting function. Frerichs, whose account of the minute appearances in fatty liver we have closely followed, further remarks, that the changes in the hepatic cells begin almost invariably at the periphery of the lobules in the province of the interlobular vessels, which contribute to the portal vein.

PATHOLOGICAL IMPORT OF FATTY LIVER.—In their discussion of the essential nature of the fatty degeneration of the liver, it is common to find it described, by different authors, as a variety of atrophy. Whilst this view of the pathological change in fatty liver has been adopted by such observers as Andral, William Thomson, Barlow, Cruveilhier, Wedl, and Heroch, they differ materially, as Frerichs has pointed out, regarding the causes of the degeneration. Frerichs admits that fatty liver sometimes commences in a disturbance of nutrition of the liver-cells ("einer Nutritionsstorung der Leberzellen"). In such instances the appearance of an excessive amount of fat in the cells is preceded by

der Leber ist, so weit meine Erfahrungen reichen, stets auf die Zellen beschränkt; niemals habe ich, auch bei den höchsten Graden dieser Veränderung Deposita von Fett in den Intercellularräumen des Parenchyms entdecken können."

[1] Fat deposited in the hepatic cells is essentially fluid; in other words, the ordinary form of the fatty matter consists chiefly of olein. Vogel, Lereboullet, and Dr. Budd describe crystallized masses, which consisted probably of margarin, as having been detected in fatty livers by the microscope; but this, according to Frerichs, is a rare appearance. Cholesterine, notwithstanding its constant existence in human bile, has never been found as an interstitial deposit in the substance of the liver. Stearin has not been found in the liver of man, but has been detected (almost to the exclusion of olein, as in Magendie's experiment) in the liver of the dog, and of other animals, when these have been fed exclusively on fatty substances.

their occupation with an abnormal plasma, and this in turn gives rise to the conversion into fat of the cell-contents. But this is not the explanation of the occurrence of fatty liver in the large proportion of cases. In such the fat is taken up into the cells from without. This reception is intimately connected with the functional activity of these structures: it increases or diminishes according to the nature of the ingesta, the richness of the blood in fat, the greater or less activity in secretion of the gland, &c. The liver acts the part of a reservoir for the surplus fat which has been absorbed, and functional derangements only occur when, owing to the overloading of the cells with fat, their other contents are compressed, and the circulation through the portal vein interfered with. When the fat has been removed from the cells by re-absorption, these damages may be removed. Such is a brief account of the view expressed by Frerichs regarding this physiological change in the liver, and which for a time, at all events, stops short of inducing really structural disease.

It is not to be lost sight of, that the presence of fat in the liver-cells is not morbid ; but that the increased or excessive amount of fat in the organ constitutes disease. Deposition of fat in the liver-cells is a natural physiological process—one which is continually at work, and the activity of which, with its subsequent re-absorption of the fatty matters is determined by the nature of the food, whether rich in fat or not, and the consequent impregnation of the blood with the same material. It is only when the character of the food has for a lengthened period been faulty, and when habits of life predisposing to excessive fatty accumulation have been long indulged in, that the process ceases to be a physiological one, and that disease is engendered.

The intimate connexion subsisting between fatty liver and tubercular diseases and the intemperate use of alcoholic drinks has already been pointed out. In these circumstances the morbid change is more apt to prove lasting, entailing those local alterations in the liver-structure to which attention has already been called, and leading to derangement of its function, and, through that, to other disturbances of the system generally.

SYMPTOMATOLOGY AND DIAGNOSIS.—Enlargement of the liver is one of the most striking features in the symptomatology of this form of disease, and one of the most important in its diagnosis ; because, although an absence of enlargement is consistent with considerable fatty degeneration of the organ, the former is infinitely the more common in its occurrence. The enlargement may be considerable, but it seldom equals the size attained by the waxy or amyloid liver, or that is sometimes reached by the carcinomatous liver. The enlargement is for the most part uniform, and the natural form of the liver is preserved. The fatty liver is less resistant to pressure, and of softer consistence, than the waxy disease. The outer surface is smooth, and the lower border usually rounded. Ascites rarely accompanies fatty

liver, neither do the superficial abdominal veins become enlarged. Jaundice is not a common symptom ; indeed, a notable degree of jaundice may be said never to occur. But the excretion of bile is interfered with, and it may become obstructed. This result is brought about mechanically, by the pressure of the distended hepatic cells on the minute ducts ; and sometimes the larger ducts share in the compression. The capillaries are likewise subjected to pressure ; and in consequence the liver in an advanced stage of fatty degeneration is always more or less anæmic. While the interference with the portal circulation is not such as to produce dropsy, it may give rise to passive congestion of the gastro-intestinal mucous surface, and so induce catarrhal affections, dyspepsia, diarrhœa, and hæmorrhoidal suffering. It rarely induces the splenic enlargements which are not uncommon in cases of cirrhosis and carcinoma.

Fatty liver is not distinguished by pain. " The organ," writes Dr. Murchison, " can be freely manipulated with impunity ; although, in extreme cases, the patient may complain of a feeling of weight or distension in the abdomen, increased by turning on the left side."[1] Reference has been made, incidentally, to a peculiar condition of the integuments observed by Dr. Addison, and believed by him to be pathognomonic of fatty degeneration of the liver. " Having," writes Dr. Addison, " in the course of my experience, been often struck with a remarkable appearance of the face in certain patients—an appearance dependent not so much on the expression of the countenance as the texture and aspect of the integuments—and having observed the exact resemblance of the appearance in each case, I endeavoured to connect it with some corresponding uniformity in the accompanying disease ; and at length arrived at the conclusion that, *when strongly marked, it is indicative, if not pathognomonic, of fatty degeneration of the liver.* It is purely integumental, as it is not confined to the face, but may pervade the whole body ; although I am disposed to think that it is earliest observable, as well as most conspicuous, in the integuments of the face and backs of the hands. To the eye, the skin presents a bloodless, almost semi-transparent, and waxy appearance : when this is associated with mere pallor, it is not very unlike fine polished ivory ; but when combined with a more sallow tinge, as is now and then the case, it more resembles a common wax model. To the touch, the general integuments, for the most part, feel smooth, loose, and often flabby ; whilst in some well-marked cases all its natural asperities would appear to be obliterated, and it becomes so exquisitely smooth and soft as to convey a sensation resembling that on handling a piece of the softest satin."[2]

In a considerable proportion of cases of fatty degeneration of the liver there will be observed a tendency, more or less marked, to fatty

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[1] Op. cit. page 45.
[2] A Collection of the published Writings of the late Thomas Addison, M.D., New Sydenham Society, page 101.

disease in other organs and tissues of the body ; and this consideration is of importance in diagnosis. If, for example, in connexion with such enlargement of the liver as is suggestive of fatty degeneration, there be present a feeble condition of the heart and pulse, a tendency to dyspnœa and to transient attacks of vertigo, and syncope, and perhaps the existence of an arcus senilis, the probability is that the heart participates with the liver in fatty degeneration.

The urine is, generally speaking, not characteristically affected in fatty liver. Should it be secreted in diminished quantity, contain albumen in greater or less amount, and deposit a sediment in which oil-casts are discovered on microscopical examination, and should a tendency to general dropsy be likewise in existence, the inference to be drawn from these circumstances is that the kidneys are also involved in the fatty transformation.

It is also to be borne in mind that the fatty degeneration of the liver is, like fatty degeneration of the heart, apt to occur in persons who manifest a tendency to take on fat. Those who lead indolent lives, partake of a too full diet, and particularly indulge freely in alcoholic drinks, are most apt to suffer.

We also look for the occurrence of fatty liver in the phthisical and the sufferers from tubercular disease in all its forms.

TREATMENT.—This will, of course, vary according to the nature of the circumstances under which the diseased condition has occurred. With a fair prospect of being serviceable, it will be adopted in those cases in which the increased amount of fat in the liver is due to a faulty arrangement of diet, provided the patients possess sufficient moral courage, and the requisite restraint, to give the required treatment fair play. By such persons, fats, starchy articles of food, sugar, malt liquors, and indeed all spirituous liquors, must be avoided ; while the lighter kinds of animal food, free from fat, green vegetables, and fruits are taken. Exercise in the open air, adapted to the strength of the individual, is to be enjoined. In prescribing a diet and regimen suitable for the removal of the fat-accumulation in the liver, great caution must be exercised, however, if there be at the same time reason to apprehend the existence of fatty degeneration of the heart.

Alkaline remedies, the alkaline carbonates or compounds of the alkalies with vegetable acids, have been found most serviceable in controlling and removing the dyspeptic symptoms so frequently associated with the liver-enlargement.

The preparations of iron are required when decided failure of strength and anæmia are apparent.

The waters of Karlsbad, Marienbad, Homburg, and Kissingen, and those of Eger in Bohemia and Ems, when a tendency to diarrhœa prevails, are recommended, in decided forms of the disease, by Frerichs.

Occurring in connexion with phthisis or other tubercular disease, the fatty condition of the liver does not demand any special modification of treatment, except that cod-liver oil and oil-inunction cannot then be regarded as suitable remedies.

The most fatty livers, as well as the largest organs, which have fallen under the writer's observation, have been in cases of chronic phthisis attended by extreme emaciation, in which cod-liver oil, either in large or moderate amount, had been daily consumed for a period of many months.

CANCER OF THE LIVER.

By J. Warburton Begbie, M.D., F.R.C.P.E.

MALIGNANT disease or Cancer of the Liver is of frequent occurrence. So much so is this the case as to justify the statement of Dr. Walshe, that "if primary and secondary cancer be taken together, it may be affirmed that no organ in the body becomes so frequently cancerous as the liver." [1]   The two most frequent of the organic diseases of the liver are cirrhosis and cancer, and of these two the latter is in all probability the more so.

It is within a comparatively recent period that Cancer of the Liver has been accurately distinguished and defined.   Formerly the disease was regarded as a consequence of hepatitis, or inflammation of the liver, and known under the name of Scirrhus.   Morgagni applied the term "Steatomata," and also the expression "Hard Tumours," to cancerous masses in the liver substance.[2]   Dr. Matthew Baillie, the well-known author of the "Morbid Anatomy," [3] employed the term "Large White Tubercles" in reference to the same masses, while incorrectly estimating their nature; and even Portal,[4] after the time when Cancer of the Liver had been recognised and fairly described by Bayle, does not mention cancer as a peculiar form of liver disease, but merely as one of the results of hepatitis.   It is to the author just named, G. L. Bayle,[5] that the merit is due of having been the first to give an accurate description of Cancer of the Liver, as well as to insist on the frequency of its occurrence.   Bayle was the earliest to demonstrate the uniform essential nature of those bodies or masses which had, antecedently to his time, been indifferently designated hard tumours, steatomata, tubercles, and white bodies, and also scirrhus, and to point out that in anatomical structure these resembled cancer of the mamma, while in the changes they underwent, and in the effects they seemed to originate in the constitution of those who presented them, they were in all important particulars similar to cancerous growths in other organs and tissues of the

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[1] The Nature and Treatment of Cancer, p. 324.
[2] De Sedibus et Causis Morborum per Anatomen indagatis, Epistola 38.
[3] Morbid Anatomy, p. 231.   5th Edition, 1818.
[4] Observations sur la Nature et le Traitement des Maladies du Foie.
[5] Dictionnaire des Sciences Médicales ; Cancer.

body. In addition to the names already mentioned, there was another, under which cancerous growths affecting the liver were long known, namely Tubera. This was first suggested by Dr. Farre; and English writers have generally followed him in using the expressions Tubera Circumscripta and Tubera Diffusa.[1] Nor are these terms to be rejected as inappropriate; on the contrary they indicate, what is very generally observed in regard to hepatic cancer, either that isolated cancerous nodules are found in the structure of the organ, or that portions more or less extensive of the hepatic tissue are infiltrated with cancer. It is in the same way and for like reason that French writers speak of "Tubercules Cancéreux" and "Tumeurs Cancéreuses Disséminées."

Cancer of the Liver is either *primary* or *secondary*. In the former case the disease is often limited to the liver, and, except to contiguous structures and organs, it rarely spreads much further. The peritoneum covering the liver, the diaphragm, duodenum, stomach, and pancreas are apt in this way to become involved. The lymphatics also suffer, and through the lymphatic vessels the disease is conducted to deep-seated glands in various parts of the body. It is not a common event for hepatic cancer to propagate itself through the veins, but secondary cancer of the lung, usually taking the form of sparsely disseminated nodules, is thus occasionally caused.

Secondary Cancer of the Liver, in the majority of instances in which it occurs, succeeds the manifestation of the disease in some other part of the portal system,—most frequently the stomach, but sometimes also the intestinal canal, the pancreas, and even the spleen. It is thus produced either through the contaminated portal blood, or through the medium of the lymphatic vessels. Cancer of the Liver may, however, result from cancerous disease in many other parts of the system. It is secondary in not a few instances to cancer of the mamma, and in such circumstances is usually associated with pulmonary cancer as well. Hepatic cancer also supervenes on the primary cancer of the uterus, ovaries, testicles, bones, skin, kidneys, and lungs. Frerichs has summed up his own observations, and those of other writers, on this head with the following results. In ninety-one cases of hepatic cancer forty-six, or one-half, were associated with carcinoma of those organs whose venous blood flows to the liver; and of these, thirty-four were cases of cancer of the stomach. Besides, among the ninety-one cases there were twenty-three instances of carcinoma in other organs, which were evidently primary, so that only twenty-two cases remain in which the liver was the organ primarily diseased. Of all the instances of hepatic cancer, then, the disease was primary in the liver in nearly one-fourth, and in three-fourths it was of a secondary nature.[2] Rokitansky acknowledges four varieties of Carcinoma of the Liver :—

1. *Areolar Cancer;* 2. *Carcinoma fasciculatum sive hyalinum* (Müller)

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[1] "It is with extreme reluctance and diffidence," writes Dr. Farre, "that the author ventures to propose another name for this disease, but the epithets *large white* are not characteristic, being common to both species, and belong, indeed, in a more remarkable degree, to species 11." Morbid Anatomy of the Liver, p. 4.

[2] Klinik der Leberkrankheiten, Band ii. S. 292.

3, 4. *Medullary carcinoma*, which occurs either as (*a*) *detached masses*, or as (*b*) an *infiltration of the hepatic parenchyma.*[1]

By Frerichs, hepatic cancer is said in general to present the characters of ordinary *simple cancer*, and according to the predominance of the fibrous element, or of the cancer juice, to belong either to the well-known *Scirrhous* or *Medullary* varieties of the disease. Other forms occasionally are met with,—for example, the *Carcinoma Melanodes*, where black pigment abounds; and the *Carcinoma Telangiectodes*, where the cancerous structure is rich in blood-vessels. Nor are these the only rare or exceptional varieties of hepatic cancer which are to be met with: tumours closely resembling sarcomatous growths (*Sarcoma*), *Cystic* and *Colloid* cancers, have been observed.[2]

Accepting the description which Frerichs has given of hepatic cancer, as at once the simplest and truest to nature, we shall now proceed to offer an account of the isolated and infiltrated forms of the disease, and in doing so shall adhere very closely to the statements of the writer just named.[3]

The isolated nodules of cancer seated in the liver vary in size from that of a millet-seed to that of an apple, or even of a child's head. In form they are for the most part roundish, but may appear flattened and even umbilicated ("nabelartig") when they extend to the outer peritoneal covering. The latter is in such circumstances opaque and thickened. There may be only one such nodule, or they may be numerous, scattered through the liver substance in its depth as well as on its surface. When large in size, the nodules are likely to be few in number. Further, they are most numerous when the hepatic cancer is secondary to new formations of a similar kind in other organs; and when themselves of primary formation, they are on the other hand more likely to remain isolated. Often, when a large cancerous tumour exists, numerous smaller ones of more recent origin are also found.

The substance of hepatic cancer is generally of a lardaceous consistence ("speckartiger Consistenz"); the mass is with far less frequency hard or gristly, and sometimes on the contrary it is soft, brain-like, almost fluctuating. On section, the surface of ordinary hepatic carcinoma presents a dull white colour, over which are visible a greater or less number of red points and streaks, according to the amount of vascularity in the mass. On pressure a milky juice is yielded, and this is always most abundant when the cancer is soft or of medullary

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[1] Dr. Walshe remarks, in regard to the varieties of cancer affecting the liver, as follows:—" Encephaloid occurs in the liver in the pure cerebriform state, and in the solanoid and hæmatoid varieties: Scirrhus, especially in the chondroid and napiform varieties: I have never seen Colloid in this organ. The scirrho-encephaloid combination is of the most frequent occurrence: next follows the solanoid variety. Fibriform stroma abounds in the scirrhous nodule. Punctiform deposition of melanic pigment is rather common: a singular shining appearance, resembling that of black granite, is thus produced in certain tumours; in others, the hue is uniformly and deeply black." Op. cit. p. 325.

[2] A Manual of Pathological Anatomy, Sydenham Society's Edition, vol. ii. p. 151.

[3] Op. cit. Band ii. S. 273.

consistence. When the cancer juice has been expressed, the network of fibres entering into the formation of the cancerous tissue is readily distinguishable. Surrounding the cancer nodules there is seldom any distinct capsule: ordinarily the new formation passes in an imperceptible manner into the surrounding liver substance. This, as well as the relation of the elements in the parenchyma of the organ to the carcinoma, is most satisfactorily determined by examining with a high magnifying power thin slices of the tumour. So doing, it is found that, in most instances, the morbid process has its commencement in the interlobular connective tissue. Important changes occur in the blood-vessels of the affected parts simultaneously with the deposition of cancerous matter in the liver substance. Just as the interlobular tissue increases, the branches of the hepatic artery become more marked, while those of the portal vein decrease. Of the latter, only isolated branches reach the cancerous mass, but of arteries, large branches pass through the fibrous framework of the cancer. Wherever the hepatic cells have given place to the cancerous elements, there is the disappearance of the capillary network formed by the portal and hepatic veins, which pertains to the normal structure of the liver, and a new vascular apparatus of abnormal arrangement is formed by the branches of the hepatic artery. There is great variety in the number of these vessels: in the milk-white tumour they are few in number, but sometimes, and more especially in soft cancers, they are sufficiently numerous to give a dark-red colour to the new formation. The walls of these vessels are for the most part thin, like the walls of capillary vessels; hence they are readily ruptured, and hæmorrhages occur. Such are usually limited to the cancer substance, but occasionally blood, owing to the giving way of the liver capsule, passes into the peritoneum. The apoplectic extravasation in liver carcinoma undergoes subsequent changes in colour, which do not however differ from those which are observed under similar circumstances in other parenchymatous tissues. When the hæmorrhages are on an extensive scale, a remarkable increase in volume occurs, and there is also sometimes anæmia produced. Sudden death from the very copiousness of the hæmorrhage may be produced when, in cases of liver cancer, the capsule of the organ is ruptured. Frerichs insists on the importance of greater attention being paid to the changes which after a length of time are produced in the vascular apparatus of the liver, more particularly in relation to the progress of fatty degeneration and softening. He believes it probable that the vessels in the cancerous depositions participate in various forms of retrograde metamorphosis. The larger branches of the portal vein, which are originally present in the liver affected by cancer, sometimes remain for a lengthened period uncontaminated, passing through the tumour, their channel free and walls healthy. This is not always the case, for not unfrequently they become filled up and obstructed by cancerous material. Cancer of the portal vein ordinarily originates from cancerous deposition in the liver; this affects the walls of the vein, and

extends from without inwards. The wall of the affected vessel becomes thickened, degenerates, and sends growths attached by a broad or narrow base into the interior of the vein, so as either partially or wholly to occlude it. Sometimes a branch of the portal vein undergoes degeneration all round in a ring-like form, and becomes obliterated by a cancerous mass closing it up. The cancer, extending from the seat of the disease in the liver structure, passes along the channel of the vein, and by degrees fills up its branches down to its capillary terminations. Thromboses more or less extensive result from cancer of the portal vein; the tumours growing from the walls of the vessel burst through its lining membrane, occasioning hæmorrhage and thrombosis. Thus, too, obstructed circulation is determined. Cancer of the portal vein is not, however, always produced in the way now described,—namely, by cancer growing from the liver into the vessel. Cases are on record in which simple venous thrombi were found to contain the elements of cancer, and in which therefore the cancer cells must have been developed in the thrombi themselves. Frerichs, Cruveilhier, and Schröder van der Kolk have all noticed the remarkable circumstance of the branches of the hepatic veins usually remaining free from cancerous infiltration.

Besides the portal vein, the lymphatic vessels, ducts, and glands are liable to be affected in Cancer of the Liver. Those of the latter lying in the fissure of the organ are specially apt to be involved, often to such an extent as to compress the bile-ducts, and obstruct the passage of bile to the duodenum, thus occasioning jaundice. Frerichs describes various changes as occurring in the bile-ducts. Large ducts not unfrequently pass unaltered through the surrounding cancerous mass. The smaller ducts are more liable to compression, and disappear just as the liver cells themselves disappear. In the hepatic and cystic ducts it is not uncommon to find cancerous nodules beneath the mucous membrane, which also lead to interruption of the bile current. When cancer exists near the surface of the liver, it is usual to find the peritoneal covering involved in the disease—a thickened condition of the peritoneum, the result of limited peritonitis, is very frequently noticed. The diaphragm and right pleura may become implicated through extension of cancer from the convex surface of the liver.

As regards the rapidity of growth of hepatic cancer, there exists very considerable variety. Soft cancers may rapidly attain increase, while scirrhous growths enlarge very slowly. On this point Dr. Walshe observes: "The enlargement of tumours in this organ (the liver) sometimes takes place with extreme rapidity, from day to day almost; whether they be primary or consecutive, a manifest increase of bulk may be detected." And again: "The duration of hepatic cancer varies extremely. There are instances in which it runs a slow and latent course for years; others in which it assumes much of the character of an acute affection."[1]

[1] Op. cit. p. 328.

Dr. Farre, in relating a case of remarkably rapid growth, observes: "A surgeon who first examined the region of the liver on the 18th of April, and again about ten days afterwards, gave it as his opinion that the liver in that short period had acquired an addition equal to at least five pounds."[1]

Changes indicative of retrograde metamorphosis and decay occur in hepatic cancer just as in the same form of disease situated in other organs and tissues of the body. These changes, according to Frerichs, consist in fatty degeneration of the carcinoma as well as in its atrophy and shrivelling-up. The cells lying in the meshes of the fibrous stroma become filled with oil-globules, and present a white opaque appearance; in this way the cancer structure assumes a reticulated aspect, or, owing to the atrophy of large groups of cells, nodular-like masses are originated. Ultimately the fatty cells may become disintegrated, and, as a consequence, an emulsive fluid may be formed, which gradually undergoes absorption. These changes occur chiefly in the central portions of the morbid growths, and the fibres of the reticulated stroma where they take place are densely aggregated, the meshes becoming smaller, until there remains only a firm cicatrized tissue, yielding on pressure no cancer juice. Dr. Hughes Bennett, after a careful examination of the preparations illustrative of the healing process of Cancer of the Liver, as described by Professor Bochdalek of Prague, concludes, "It is very probable, therefore, that cancer may undergo transformations, sometimes fatty, and at other times calcareous; that its power of growth and re-development may be checked, and as a consequence it shrivels up, some of the softer parts are absorbed, and the remainder continues inert in the system, while the contraction of the surrounding parts, and the filamentous stroma of the cancer together, constitute the puckerings and cicatrices occasionally found as evidences of a spontaneous cure."[2] The same author has still more strongly expressed his belief in the spontaneous cure of cancer. He remarks (page 215), "The facts recorded in the first part of the work, I think, afford unequivocal proof that a cancerous growth may undergo spontaneous cure; and I feel persuaded that when evidences of this result are more diligently sought after than they have hitherto been, it will be found to have occurred much more frequently than is generally supposed." Frerichs, on the other hand, doubts the conclusion to which their observations have led Bochdalek, Oppolzer, and Hughes Bennett, regarding the retrograde metamorphosis already described as by no means significant of actual cure in cancerous disease;[3] and he has offered the very important suggestion that Oppolzer and Bochdalek may have confounded carcinoma with the syphilitic cicatrices of the liver, the nature of which is now much better understood than formerly. In defending his position, Frerichs

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[1] Op. cit. p. 28.   [2] On Cancerous and Cancroid Growths, p. 212.
[3] Frerichs emphatically observes: "Einen Fall von wahrer Heilung des Leberkrebses habe ich weder am Krankenbette, noch am Leichentische jemals constatiren können." Op. cit. p. 283.

has also stated that, although the retrograde change may be distinguished in the centre of the cancerous nodule, a progressive development of disease is observed at the circumference; and from this he concludes that the morbid action has not expired, but is only locally destroyed.

Reference has already been made to the varieties of hepatic cancer, the *scirrhous* and *medullary* having been signalized as the two most common in their occurrence. It is to these that the preceding description applies. Besides the scirrhous and medullary, Frerichs has especially indicated the four following varieties:—1. Fungus Hæmatodes. 2. Melanotic Cancer. 3. Cystic Cancer. 4. Alveolar, or Colloid Cancer. A few remarks may now be made regarding these, in the order named.

1. *Fungus Hæmatodes.* To this form of cancer, abounding as it does in blood-vessels, the term "Carcinoma Telangiectodes" is also applied. The vessels themselves are of large size, and possess thin walls; they are therefore readily torn, and extravasations of blood are occasioned. Instances of this peculiar form of disease are rare; but there exists a still rarer variety included under the head fungus, in which certain portions of the cancer consist of creatile tissue. Plate IV. in Dr. Farre's work[1] is illustrative of the fungous character of the "Tubera Diffusa," Fig. II. being a representation of "the external and internal appearances of the tubera in their different stages, and the ultimate stage is shown by the largest tuber, which had destroyed the peritoneal tissue of the liver, and protruded in the form of a bleeding fungus."

2. *Melanotic Cancer.* This variety of hepatic cancer is characterised by the dissemination through the liver substance of small nodules, presenting in part a pale, and in part a yellow, brown, or black pigmentation.[2] The observation of Dr. Walshe, as to the "rather common" occurrence of punctiform deposition of melanic pigment, has already been referred to. Both Walshe and Frerichs describe the appearance of the liver so affected as in some instances resembling that of granite.[3] The pigmentary deposit takes place, generally speaking, in the interior of the cancer cells, but is also found in the form of isolated or aggregated granules in the cancer juice. Melanotic cancer tends to grow rapidly.[4]

3. *Cystic Cancer.* Under the name of Carcinoma Cysticum ("der Cystenkrebs") Frerichs describes a form of Cancer of the Liver characterised by the presence of round holes, from the size of a pea to that of a walnut, filled with a clear serous fluid, and lined by a smooth

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[1] Op. cit. page 52.

[2] The colour of melanotic tumours of the liver varies, according to the quantity of pigment granules they contain. In the same liver, tumours may sometimes be found of every shade, from light brown to black. Budd, Diseases of the Liver, p. 301.

[3] "Das organ erhält dadurch ein granitähnliches Aussehen." Frerichs.

[4] Medullary cancer not unfrequently occurs in the liver in the shape of cancer melanodes (melanosis), and equally as an infiltration, or in circumscribed masses. Rokitkansky, op. cit. vol. ii. p. 154.

membrane, resembling a serous membrane. These are found in the
cancerous nodules. Not unfrequently, the border of the hole is in-
distinct, and the serous membrane absent, while the contents, instead
of being clear, consist of a slimy, viscid fluid.

4. *Colloid, or Alveolar Cancer.* This is, unquestionably, a very rare
form of hepatic cancer. Dr. Budd remarks, " Every variety of cancer,
excepting, perhaps, gelatiniform, or colloid cancer, has been met with
in this organ."[1] Dr. Walshe writes, " I have never seen colloid in
this organ."[2] Nevertheless, colloid of the liver is occasionally met with.
Frerichs has found it once, and Van der Byl, in the " Transactions of
the Pathological Society of London,"[3] has recorded another instance.

SYMPTOMATOLOGY.—Cancer of the Liver, whether occurring in its
ordinary condition of scirrhous and medullary, or in one of the rarer
forms now briefly alluded to, occasions an increase in the size of the
organ. That increase is sometimes very great. A cancerous liver has
been known to weigh fifteen pounds. The greater part of the abdominal
cavity may be occupied by a liver thus affected. The enlargement
takes place progressively, and often, particularly in the softer forms of
the disease, with rapidity. It is of importance, however, to keep in
view that, although augmented size is characteristic of hepatic cancer,
the enlargement may be so slight as to escape detection during life.
The organ may originally have been a small one, and the presence of
cancerous deposition may not have caused it to project beyond the
ribs, or a distended bowel may have overlapped the lower margin.
Dr. Murchison refers to an instance of this kind, in which, unex-
pectedly, the presence of large cancerous nodules was found on post-
mortem examination.[4] The enlargement is very generally irregular,
owing to the presence of nodular excrescences. These, when well
marked, as they often are, greatly facilitate diagnosis. They are to be
felt by the hand over the surface of the enlarged organ, or at its lower
margin, and not uncommonly in the advanced stages of the disease,
when the patient is greatly emaciated, they are readily recognised by
sight. So notable is the irregularity of the enlarged cancerous liver,
that in some instances the tumour affects one portion of the organ
only. Dr. Bright has recorded a remarkable example of malignant
tumour confined entirely to the left lobe of the liver, and ascending
towards the thorax.[5] It was situated in the left hypochondriac region,
and originated within the left lobe of the liver, which pushed the stomach
to the right side. The tumour within the liver was of the size of an
adult's head, and of a rounded form ; its external surface was firmly
adherent to portions of the lower surface of the diaphragm, and pos-
teriorly to the spleen and kidney. On cutting into the tumour. it

[1] Op. cit. p. 391.   [2] Op. cit. p. 325.   [3] Vol. ix.
[4] Clinical Lectures on Diseases of the Liver, p. 188.
[5] Clinical Memoirs on Abdominal Tumours and Intumescence, New Sydenham
Society, Case 10, p. 260. There are other instances of a similar nature recorded in Dr.
Bright's Reports, also in Andral's Clinique Médicale ; and Dr. Murchison, op. cit. p. 188,
refers to one which had fallen under his own observation.

was found to be of a fungoid nature (fungus hæmatodes), originating within the structure of the left lobe of the liver: internally, it was in some parts rather soft, of a dark red colour, resembling a clot of extravasated blood ; whilst in others its structure was of a pale colour, resembling cerebriform cancer. In other places, the surface of the liver presented a hobnailed appearance : it also contained a small portion of fat."

The enlargement of the liver determined by cancer is essentially painful. The patient complains of great uneasiness, and when pressure over the enlarged organ is made, the tenderness and pain experienced are apt to be great, the latter radiating in the direction of the shoulders and back. Jaundice is a variable symptom in hepatic cancer, and it is perhaps more frequently absent than present. When jaundice has once appeared in a case of Cancer of the Liver, it never disappears. Dr. Walshe remarks, "Jaundice, without ascites, occurs in somewhat less than half the cases, and, so far as I have observed, only appears under the influence of pressure directly exercised on the large biliary ducts ; it is hence often connected with infiltration of the lesser omentum."[1] That jaundice is generally produced by the direct compression of the bile-duct by a cancerous nodule in the liver, or by enlarged glands in the portal fissure (Murchison), is no doubt true; and it is equally true that, without such pressure, the whole secreting tissue of the liver may be destroyed, and no jaundice result; but instances have been recorded in which no such obstruction has existed, and no appreciable lesion of the bile-ducts has been detected. Andral more especially has detailed such cases.[2] Ascites, like jaundice, is a variable symptom in Cancer of the Liver; probably it occurs a little more frequently than jaundice, although Dr. Walshe states the relative frequency otherwise.[3] Frerichs, in eighteen out of thirty-one cases,

[1] Frerichs remarks regarding jaundice :—" As a symptom of carcinoma of the liver, jaundice is of little value ; in the majority of cases it does not exist : it was only present in thirteen out of thirty-one falling under my own observation, and in twenty-six out of sixty noticed by other physicians. Thus, fifty-two out of ninety-six cases did not present this symptom."

[2] Clinique Médicale ; Maladies de l'Abdomen, Quatrième Partie, p. 274. The thirty-fifth observation, more particularly, is one in point. "Cancer du foie et de la rate. Tumeur douloureuse dans l'hypocondre droit. Ictère. Pas d'ascite. Ramollissement de la membrane muqueuse gastrique. Couleur ardoisée du duodenum." In giving an account of the post-mortem appearances in this case, Andral observes, "Aucune lesion appréciable ne fut trouvée dans les canaux hépatique, cholédoque et cystique, non plus que dans la vésicule : celle-ci contenait, au lieu de bile liquide, plusieurs petits calculs unis entre eux par du mucus."

[3] " Ascites without jaundice is a much less frequent consequence of the disease. Jaundice and ascites combined occur in a fourth or fifth of all cases ; in about a third of the whole number neither of these symptoms is present. Either of them may be the sole existing symptom of hepatic disease : jaundice, however, as just signified, much more frequently than ascites." Op. cit. p. 329. The possible explanation of the different opinions expressed by different writers respecting the occurrence of jaundice in cancer of the liver depends on the circumstance that some may have only considered jaundice to exist when its presence was notable—as declared by a yellow colour of the surface of the body, bile pigment in the urine, and clay-coloured stools ; whereas the simple colouring of the conjunctivæ, with a doubtful yellow tint of the cutaneous surface, may have been considered jaundice by others.

found a large accumulation of fluid ("grössere Mengen von Fluidum") in the peritoneal sac; in five of these the fluid was clear serum, in eight sero-fibrinous, in four bloody, and in one pure blood. In sixty instances recorded by different authors, and referred to by Frerichs, ascites existed in thirty, in nineteen it was absent, and in the remaining eleven there is no notice of its existence or absence taken. Nearly all observers are agreed in attributing the ascites, for the most part, to an affection of the peritoneum, or chronic peritonitis, which commences over the liver surface. Frerichs styles this the usual cause. Dr. Murchison remarks : "The fluid may be a simple dropsical collection, due to compression or obstruction with cancerous matter of the trunk or large branches of the portal vein, but the amount is usually small as compared with what is observed in cirrhosis. Oftener it is the result of a chronic peritonitis originating on the surface of the liver." Dr. Walshe observes that, "When slight in amount, the accumulation of fluid appears to result from peritoneal irritation; when abundant, from pressure of the large venous trunks." The amount of fluid is generally not large, but occasionally it does become very great; and sometimes the rapidity of its collection is remarkable, notably exceeding the rapidity with which ascitic fluid gathers in cases of cirrhosis, a lesion much more frequently occasioning abdominal dropsy than hepatic cancer does. Hydræmia cannot be said to determine the ascites, but it probably favours the operation of the true causes, which are those now referred to. Œdema of the dependent portions of the body bears to Cancer of the Liver no fixed or definite relationship, but it is not unfrequently observed in the advanced stages of some cases, these especially characterised by the existence of the tumid belly with considerable or great ascites. Where very decided obstruction to the portal circulation exists—and this, as already stated, is exceptional as compared with cirrhosis—the superficial abdominal veins are apt to become enlarged and turgid, as is so commonly noticed in the latter form of hepatic disease. Enlargement of the spleen, so marked a feature in cirrhosis, and in some other diseases of the liver, is of infrequent occurrence in cancer. Of ninety-one cases referred to by Frerichs, splenic tumour existed only in twelve; in all the others the organ was either normal in size, or smaller than usual.

The urine in Cancer of the Liver will vary, accordingly as jaundice is or is not present. In the former case, bile pigment will impart its own peculiar tint to the secretion. Notably, however, the urine in Cancer of the Liver is scanty and loaded with pigment. In no other form of hepatic or of pulmonary disease, with which the carmine or vermilion-coloured deposit of urates is so intimately associated, has the writer had occasion to notice such large and persistent presence of these deposits as in certain instances of hepatic cancer.[1] But, while

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[1] Becquerel, in treating of the urine in Cancer of the Liver, observes, "Un des effets les plus remarquables des maladies organiques du foie est de produire les urines febriles au plus haut degré : quantité peu considérable, forte coloration, forte densité, sédiments abondants, rougeâtre et briquetés d'acide urique." Sémeiotique des Urines, p. 415.

the diminished secretion and high colouration of the urine distinguish all cases of hepatic cancer at an early stage, and many cases throughout their entire progress, it is also noticeable that, when the patient has become much exhausted by pain, insufficient nourishment, and it may be by diarrhœa or hæmorrhage, the urine ceases to present the febrile character, and acquires the anaemic ("urine anémique" of Becquerel). This has been noticed by Dr. Walshe,[1] and is also referred to by Dr. Parkes.[2]

In the early stages of the disease, the symptoms which usually present themselves are those of depraved digestion, anorexia, nausea, vomiting, constipation, and flatulent distension, with eructations of gas, which is often fetid. To these succeed pain in the region of the liver, frequently a short dry cough, and an irregular condition of the bowels, the constipation alternating with diarrhœa. Many of the more prominent symptoms of hepatic cancer are those also of cancer of the stomach, and it need hardly be remarked, that the former are always greatly aggravated by the co-existence of the latter disease.

As the disease advances, and the enlargement of the liver becomes extreme, uneasiness in the præcordia, dyspnœa, and disturbed action of the heart are apt to be induced. The emaciation resulting from this disease is frequently excessive, and more or less attenuation is the ordinary condition of the sufferers from hepatic cancer in its advanced stages. Exceptions to this rule have, however, been noticed. Frerichs and Oppolzer have observed cases of medullary cancer attended by no emaciation; the patients, on the contrary, presenting a plump appearance ("wohlgenährt"). Sudden death may occur, owing to the rupture of a cancerous tumour into the cavity of the peritoneum, or from perforation of the diaphragm (Cruveilhier). An attack of acute peritonitis, or of pleurisy, resulting from the irritation, or from the actual extension to these membranes of the cancerous disease, may, on the other hand, swiftly terminate life. Still more common, however, is it for the sufferer from hepatic cancer to perish gradually, the termination being that of the cachectic condition into which he has lapsed, from the entire failure of the digestive and assimilating functions.

There exists unquestionably variety in the duration of hepatic cancer. The disease generally, although not invariably, runs a rapid course. The progress of medullary carcinoma is swifter than that of scirrhus. A period from a few weeks to a few months, rarely exceeding twelve months, probably includes the duration of this mortal malady in whatever form it occurs, and the mean duration of it may, with equal probability, be fixed at six months. Dr. Budd, Dr. Walshe, and Frerichs, it is true, assign a longer period to scirrhus: Dr.

[1] Op. cit. pp. 117 and 330.

[2] "Sometimes, and especially in the last stages, the urine is copious, pale, and deficient in urea. This has evidently depended, in my cases, in great measure on the utter failure of digestive and nutritive power. In a woman with enormous hepatic cancer and profuse diarrhœa A. Vogel found the urine very copious, with little pigment, &c.; in each twenty-four hours there were only 122 grains of urea." The Composition of the Urine, p. 330.

Budd stating that "life may be prolonged to a year and a half or two years;" Dr. Walshe remarking that "there are instances in which it runs a slow and almost latent course for years;" and Frerichs, that "the disease may last for years:" but it is nevertheless true, that Cancer of the Liver is a disease which runs a rapid course, and that the following observation of Dr. Murchison is justified by extended observation and experience:—" The very fact of an enlargement of the liver having lasted much longer (than a twelvemonth) would be an argument against its being due to cancer." [1]

ETIOLOGICAL CONSIDERATIONS.—The influence of *age* is important. Primary Cancer of the Liver is extremely rare before adult age. It may be said to occur more frequently between the ages of fifty and seventy. Two-thirds of the total number of deaths from hepatic cancer take place between fifty and seventy (Walshe). Of eighty other cases, including thirty-one observed by Frerichs himself, and fifty-two recorded by other authors, there occurred seven between twenty and thirty years of age, fourteen between thirty and forty, forty-one between forty and sixty, nineteen between sixty and seventy, and two above seventy, or nearly one-half between forty and sixty. In twenty-nine cases recorded by Van der Byl ("Transactions of Pathological Society of London," vol. ix.), the mean age of the males (thirteen in number) was forty-one, and that of the females (sixteen in number) fifty. The youngest individuals affected with hepatic cancer, who have fallen under the observation of Frerichs, were a young man and young woman, of twenty and twenty-two years of age respectively. In both, the disease was secondary: in the former, there had existed medullary fungus of the testes; in the latter, cancer of the eyeball, on account of which it had been extirpated. Very much younger subjects than those of Frerichs' sufferers from secondary cancer of the liver have been observed. Farre, for example, records in detail the following cases:—1. A boy of two years and seven months had an enlarged testicle, which proved to be cancerous, and to which tumours of the liver and of the lung succeeded. 2. An infant three months old had a tumour originating behind the peritoneum, and enveloping the left kidney, to which Cancer of the Liver was secondary. 3. A boy, two years and six months old, had a tumour at the outlet of the pelvis, near the anus, and to this tubera of the liver succeeded, while the alimentary canal, the mesenteric glands, the thoracic duct, kidneys, spleen, and pancreas remained unaffected. [2] Persons of the most advanced age sometimes become the victims of Cancer of the Liver (Walshe, Budd).

Sex, in all probability, exerts little or no influence as a predisposing cause of hepatic cancer. Of one hundred and sixty-five cases, collected from different authors, the writer finds eighty-five to have been females, and eighty males. Dr. Walshe's statement would imply that males are more commonly affected than females.

[1] Op. cit. p. 192.   [2] Op. cit. p. 38.

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of the conjunctivæ, with a doubtful yellow tint of the cutaneous surface, may b
considered jaundice by others.

found a large accumulation of fluid (" grössere Mengen von Fluidum") in the peritoneal sac; in five of these the fluid was clear serum, in eight sero-fibrinous, in four bloody, and in one pure blood. In sixty instances recorded by different authors, and referred to by Frerichs, ascites existed in thirty, in nineteen it was absent, and in the remaining eleven there is no notice of its existence or absence taken. Nearly all observers are agreed in attributing the ascites, for the most part, to an affection of the peritoneum, or chronic peritonitis, which commences over the liver surface. Frerichs styles this the usual cause. Dr. Murchison remarks : " The fluid may be a simple dropsical collection, due to compression or obstruction with cancerous matter of the trunk or large branches of the portal vein, but the amount is usually small as compared with what is observed in cirrhosis. Oftener it is the result of a chronic peritonitis originating on the surface of the liver." Dr. Walshe observes that, "When slight in amount, the accumulation of fluid appears to result from peritoneal irritation; when abundant, from pressure of the large venous trunks." The amount of fluid is generally large, but occasionally it does become very great; and sometimes the rapidity of its collection is remarkable, notably exceeding the rapidity with which ascitic fluid gathers in cases of cirrhosis, a lesion much more frequently occasioning abdominal dropsy than hepatic cancer does. Hydræmia cannot be said to determine the ascites, but probably favours the operation of the true causes, which are those referred to. Œdema of the dependent portions of the body bears Cancer of the Liver no fixed or definite relationship, but it is not infrequently observed in the advanced stages of some cases, these specially characterised by the existence of the tumid belly with considerable or great ascites. Where very decided obstruction to the portal circulation exists—and this, as already stated, is exceptional as compared with cirrhosis—the superficial abdominal veins are apt to become enlarged and turgid, as is so commonly noticed in the latter form of hepatic disease. Enlargement of the spleen, so marked a feature in cirrhosis, and in some other diseases of the liver, is of infrequent occurrence in cancer. Of ninety-one cases referred to by Frerichs, splenic tumour existed only in twelve; in all the others the organ was either normal in size, or smaller than usual.

The urine in Cancer of the Liver will vary, accordingly as jaundice is or is not present. In the former case, bile pigment will impart its own peculiar tint to the secretion. Notably, however, the urine in Cancer of the Liver is scanty and loaded with pigment. In no other form of hepatic or of pulmonary disease, with which the carmine or vermilion-coloured deposit of urates is so intimately associated, has the writer had occasion to notice such large and persistent presence of such deposits as in certain instances of hepatic cancer.[1] But, while

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Becquerel, in treating of the urine in Cancer of the Liver, observes, "Un des effets remarquables des maladies organiques du foie est de produire les urines febriles au degré : quantité peu considérable, forte coloration, forte densité, sédiments rougeâtre et briquetés d'acide urique." Séméiotique des Urines, p. 415.

the diminished secretion and high colouration of the urine
all cases of hepatic cancer at an early stage, and many case
out their entire progress, it is also noticeable that, when t
has become much exhausted by pain, insufficient nourishm
may be by diarrhœa or hæmorrhage, the urine ceases to
febrile character, and acquires the anæmic ("urine ané
Becquerel). This has been noticed by Dr. Walshe,[1] and
ferred to by Dr. Parkes.[2]

In the early stages of the disease, the symptoms which u
sent themselves are those of depraved digestion, anorex
vomiting, constipation, and flatulent distension, with eru
gas, which is often fetid. To these succeed pain in the reg
liver, frequently a short dry cough, and an irregular condi
bowels, the constipation alternating with diarrhœa. M
more prominent symptoms of hepatic cancer are those also
of the stomach, and it need hardly be remarked, that the
always greatly aggravated by the co-existence of the latter

As the disease advances, and the enlargement of the live
extreme, uneasiness in the præcordia, dyspnœa, and distur
of the heart are apt to be induced. The emaciation resultin
disease is frequently excessive, and more or less attenuation i
nary condition of the sufferers from hepatic cancer in its
stages. Exceptions to this rule have, however, been noticed
and Oppolzer have observed cases of medullary cancer atter
emaciation; the patients, on the contrary, presenting a plur
ance ("wohlgenährt"). Sudden death may occur, owing to t
of a cancerous tumour into the cavity of the peritoneum, or
foration of the diaphragm (Cruveilhier). An attack of acute
or of pleurisy, resulting from the irritation, or from the act
sion to these membranes of the cancerous disease, may, or
hand, swiftly terminate life. Still more common, however, i
sufferer from hepatic cancer to perish gradually, the termina
that of the cachectic condition into which he has lapsed
entire failure of the digestive and assimilating functions.

There exists unquestionably variety in the duration
cancer. The disease generally, although not invariably, ru
course. The progress of medullary carcinoma is swifter th
scirrhus. A period from a few weeks to a few months,
ceeding twelve months, probably includes the duration of t
malady in whatever form it occurs, and the mean dura
may, with equal probability, be fixed at six months. Dr.
Walshe, and Frerichs, it is true, assign a longer period to sci

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[1] Op. cit. pp. 117 and 330.
[2] "Sometimes, and especially in the last stages, the urine is copious,
ficient in urea. This has evidently depended, in my cases, in great measur
failure of digestive and nutritive power. In a woman with enormous h
and profuse diarrhœa A. Vogel found the urine very copious, with little p
in each twenty-four hours there were only 122 grains of urea." The Comp
Urine, p. 330.

Budd stating that "life may be prolonged to a year and a half or two years;" Dr. Walshe remarking that "there are instances in which it runs a slow and almost latent course for years;" and Frerichs, that "the disease may last for years:" but it is nevertheless true, that Cancer of the Liver is a disease which runs a rapid course, and that the following observation of Dr. Murchison is justified by extended observation and experience :—" The very fact of an enlargement of the liver having lasted much longer (than a twelvemonth) would be an argument against its being due to cancer."[1]

ETIOLOGICAL CONSIDERATIONS.—The influence of *age* is important. Primary Cancer of the Liver is extremely rare before adult age. It may be said to occur more frequently between the ages of fifty and seventy. Two-thirds of the total number of deaths from hepatic cancer take place between fifty and seventy (Walshe). Of eighty other cases, including thirty-one observed by Frerichs himself, and fifty-two recorded by other authors, there occurred seven between twenty and thirty years of age, fourteen between thirty and forty, forty-one between forty and sixty, nineteen between sixty and seventy, and two above seventy, or nearly one-half between forty and sixty. In twenty-nine cases recorded by Van der Byl (" Transactions of Pathological Society of London," vol. ix.), the mean age of the males (thirteen in number) was forty-one, and that of the females (sixteen in number) fifty. The youngest individuals affected with hepatic cancer, who have fallen under the observation of Frerichs, were a young man and young woman, of twenty and twenty-two years of age respectively. In both, the disease was secondary : in the former, there had existed medullary fungus of the testes; in the latter, cancer of the eyeball, on account of which it had been extirpated. Very much younger subjects than those of Frerichs' sufferers from secondary cancer of the liver have been observed. Farre, for example, records in detail the following cases :—1. A boy of two years and seven months had an enlarged testicle, which proved to be cancerous, and to which tumours of the liver and of the lung succeeded. 2. An infant three months old had a tumour originating behind the peritoneum, and enveloping the left kidney, to which Cancer of the Liver was secondary. 3. A boy, two years and six months old, had a tumour at the outlet of the pelvis, near the anus, and to this tubera of the liver succeeded, while the alimentary canal, the mesenteric glands, the thoracic duct, kidneys, spleen, and pancreas remained unaffected.[2] Persons of the most advanced age sometimes become the victims of Cancer of the Liver (Walshe, Budd).

Sex, in all probability, exerts little or no influence as a predisposing cause of hepatic cancer. Of one hundred and sixty-five cases, collected from different authors, the writer finds eighty-five to have been females, and eighty males. Dr. Walshe's statement would imply that males are more commonly affected than females.

[1] Op. cit. p. 192.   [2] Op. cit. p. 38.

Dr. Budd's assertion, that "we have no evidence that (
the Liver is more frequent in hot climates than in our o'
persons who drink spirits to excess than in those who abs
them," is amply comfirmed by the experience of other writ
hereditary transmission of cancer is almost universally
but sufficient data do not exist for determining whethe
Cancer of the Liver depends in any notable degree upon l
taint.

Diagnosis.—Although, under ordinary circumstances, the re
of Cancer of the Liver is by no means difficult, yet cases every
then do present themselves, in which the diagnosis is rer
from easy. When, as usually happens, after the disease has e
some time, the liver is enlarged in size, and the prominences on
can be distinguished on palpation, there is probably no form (
malady which can be more readily or certainly diagnosed. B
already been stated, the liver may be the seat of cancer, wit
enlargement having occurred, and in such circumstances we
be led to a limited extent towards correct diagnosis, from no
emaciation of the patient, together with the symptoms of in
which are, at all events, likely to be present. Frerichs has
that in similar instances the invariable tenderness which tl
experiences when percussion over the liver is practised,
mencing ascites, and the absence of any other cause for the
condition, will afford grounds for the suspicion that Cancer of
exists, although, he adds, a certain opinion in such circum
not possible.

It behoves us also to bear in remembrance that, at its comm
and during its early progress, great obscurity surrounds the
inasmuch as the decided enlargement and the prominent
the surface, the only unquestionable indications of cancer,
advanced stage of the disease.

There are, moreover, certain other forms of hepatic dise
certain diseases of neighbouring organs, which may be mis
cancer, and with which, on the other hand, cancer may be co
Frerichs refers to ten such. These are, in the order named b
1. Waxy liver.[1] 2. Syphilitic hepatitis. 3. Liver depres
habit of tight lacing ("Schnürleber"). 4. Hydatid disease
5. Hepatic abscesses. 6. Dilatation of the bile-ducts and gal
resulting from stoppage of the hepatic and choledic ducts.
of the omentum. 8. Cancer of the stomach. 9. Cancer of
kidney. 10. Accumulation of feculent masses in the transve
To these ten another may be added, namely, the cirrhotic li
great distension of the peritoneum, by ascitic fluid, wholly

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[1] It is especially in the cases of combined waxy liver and cirrhosis ("der c
Wachsleber") that the diagnosis is apt to be difficult. A nodular surface ex
this form of disease and cancer, but the nodules in the former case are m
than they are in carcinoma.

the reliable determination of the size of the liver. No doubt, the increased dimensions of the liver in the case of carcinoma, even when accompanied by very considerable ascites, are, generally speaking, to be readily enough ascertained, but from time to time examples occur of cachectic-looking patients, who have passed the meridian of life, have become much emaciated, have slight jaundice, tenderness over the hepatic region, possibly no splenic enlargement, and urine which is equally characteristic of cancer and cirrhosis. In such it is a matter of great difficulty, without having recourse to paracentesis abdominis, to determine the real nature of the disease; and it is certainly necessary to be very cautious in reaching a diagnosis, for in precisely such circumstances the most careful and observant physicians have fallen into error. It seems unnecessary to enter into any detailed account of the salient points in the differential diagnosis between cancer on the one hand and the various diseases which have now been named; these are all signalized in the separate description of the affections themselves.

PROGNOSIS.—Cancer of the Liver is a uniformly fatal disorder; the prognosis therefore, when once an accurate diagnosis has been formed, is necessarily of the worst possible description. It has already been stated, that a certain variety exists in respect to the rapidity with which the disease advances in different cases—the scirrhous cancer being, as a general rule, less speedy than the medullary. A knowledge of this well-ascertained circumstance should be permitted to modify the opinion as to duration of the disease which the physician may be called upon to express.

TREATMENT.—The only opportunity afforded is in the way of palliative treatment; for the relief, or it may be the removal, of various distressing symptoms, something at least may be done. The rule " ne nimia diligentia" is applicable to cases of Cancer of the Liver, supposing an accurate diagnosis of the disease to have been made. Anything like active treatment is wholly inadmissible. Mercury, which has for so long a time been invariably prescribed in disorders of the liver, can, in this disease, only do harm, and has been known to hurry on, with great rapidity, the occurrence of the fatal event. Frerichs expresses himself in nearly similar terms in regard to the use of iodine, arsenic, and the Karlsbad waters. It is true that Dr. Walshe has stated that the progress of the affection has appeared to have been stayed by the liberal inunction of the iodide of lead ointment over the hepatic region, and the internal administration of liquor potassæ in infusion of taraxacum.

It may be affirmed, without any fear of contradiction, that the great objects for the physician to keep in view, in the treatment of cases of hepatic cancer, are fourfold:—1. To support the patient's strength by the due administration of suitable food. 2. The promotion

of digestion. 3. The relief of pain; and lastly, The procuring of refreshing sleep.

Diet must be light, and at the same time nutritious; nitrogenized much more than carbonized. Saccharine, oily, and starchy substances are to be avoided, as calculated to throw increased duty on the liver; and on the other hand, the lean of animal food, and carefully prepared soups, with well-boiled green vegetables, are to be administered. Food should be taken in small quantities and frequently, rather than in larger amount and after rarer intervals. Alcoholic stimulants, which, sooner or later, will be required, ought always to be given freely diluted with water, and in as moderate doses as possible.

There are various remedies, which the exigencies of each individual case will suggest, for the relief of the frequently disturbing and often very annoying dyspeptic symptoms which are associated with hepatic cancer. Of antacids, bismuth and magnesia are the chief; while for the alleviation of sickness and vomiting, in the former, with or without a little hydrocyanic acid, in the acid itself, and in ice, we find the most reliable remedies. Belloc's charcoal is an invaluable adjuvant when flatulence and its attendant distension exist; and for the same purpose Dr. Murchison suggests from fifteen to thirty minims of a saturated aqueous solution of carbolic acid with a few drops of chloric ether in peppermint water. Gentle laxative enemata should be regularly employed for the relief of the bowels, and this means is to be considered as decidedly preferable to the use of any laxative medicine, unless of the mildest description, by the mouth. The writer has found the phosphate of soda, either introduced into such articles of diet as soup, or simply dissolved in tepid water, to be a safe and, generally speaking, certain laxative. He can also recommend the use, under similar circumstances, of Kurella's powder, the pulvis glycyrrhizæ compositus of the Prussian Pharmacopœia, in one or two drachm doses, mixed with water or treacle.

As anodynes and hypnotics there are, of course, many remedies available, but it may be confidently affirmed that in no disease is the soothing influence of opium, in one or other of its various preparations, more frequently called for, and in none do these apparently act with greater advantage and with fewer drawbacks than in the painful diseases of the liver, and perhaps chiefly in cancer of this organ. It is unnecessary to specify the different forms of the drug which may be employed, seeing that one preparation acts best at one time, and another preparation at another, even in the same case. It is a good rule, however, to begin with the administration of as small a dose as is consistent with securing the desired soothing or anodyne impression.

With an excessive increase of the ascites, as sometimes occurs, the breathing is rendered very difficult, and it may be necessary to perform paracentesis. This operation should only be had recourse to when the dyspnœa has become urgent, as it is rarely, if ever, succeeded

by more than merely temporary relief, while, on the other hand, the re-accumulation of fluid in the peritoneal cavity occurs with increased rapidity after its performance, and the strength of the patient also rapidly deteriorates. When fluid has collected to any extent, it is very seldom re-absorbed ; and on this account the employment of drastic purgatives, which are often useful in the treatment of ascites dependent on other causes, is in this disease to be avoided.

HYDATID DISEASE OF THE LIVER.

By J. WARBURTON BEGBIE, M.D., F.R.C.P.E.

THE term hydatid (*ὑδατίς*, a vesicle, from *ὕδωρ*, water) was employed by some of the ancient physicians; and the authors referred to, although unaware of its real nature, were evidently familiar with a form of disease of the liver closely resembling that which is now known under the name of Hydatids. Hippocrates, for example, has the following—"Ὁκόσοισι δ' ἂν τὸ ἧπαρ ὕδατος πλησθὲν ἐς τὸ ἐπίπλοον ῥαγῇ, τούτοισι ἡ κοιλίη ὕδατος ἐμπίπλαται καὶ ἀποθνήσκουσι:"[1] and Aretæus, in describing the different forms of dropsy, relates that " Small and numerous bladders, full of fluid (" *κύστιες σμικραὶ, συχναὶ, πλήρεες ὑγροῦ*"), are contained in the place where ascites is found; but they also float in a copious fluid. . . . . . It is said, however, that in certain cases such bladders have come out by the bowels."[2] Many observers in the sixteenth and seventeenth centuries have recorded facts which indicate their acquaintance with hydatid tumours of the liver. Cristophorus de Vega,[3] who flourished about the middle of the earlier century, remarks: " Vidimus sæpe jecur, non in nobis tantum sed in animalibus occisis, plenum aqua, quoniam in membrana ipsum obvolvente continetur, plures efficiens vesiculas : hæ quoque rumpuntur." And Felix Plater, the earliest nosologist, whose works were first published at the commencement of the seventeenth century, observes: " Vesiculas tenuissimas pellucidas aqua distentas, pomi magnitudinem nonnunquam æquantes, hepatis substantiæ accrevisse, in cachecticis sæpe inveni: sed similes ex hepati et liene simiæ . . . excepi."[4] In the Sepulchretum of Bonetus (lib. iii. sect. xxi.) a case is recorded by Riverius, the interest of which is increased by the satisfactory nature of the cure : " Rusticus quidam hydropicus factus abscessum passus est in dextra parte abdominis; eoque aperto, infinitus prope modum vesicularum aqua repletarum numerus egressus est, ut ducentorum numerum excederet, idque per plurium dierum spatium, et sic omnino curatus est." Besides these, there were many other observations made ; but, notwithstanding this circumstance, the real nature of the

[1] ᾽ΑΦΟΡΙΣΜΟΙ᾽. ΤΜΗ͂ΜΑ ῞ΕΒΔΟΜΟΝ. Α. νέ. [2] ΧΡΟΝΙΩΝ ΠΑΘΩΝ. ΒΙΒΛ᾽. Β.
[3] Cristophorus de Vega. Opera omnia. Leon. 1586.
[4] Felix Pater. Praxis Medica. Basil, 1602-4.

hydatid tumour remained for a lengthened period unrecognised. To use the words of Davaine :[1] " Mais aucun des auteurs ou des observateurs antérieurs à Pallas (1766, 1767) ne soupçonna que ces vésicules jouissent d'une vie indépendante." Before Pallas wrote in 1760 (" De infestivis Viventibus intra Viventia." East in Thesaur. Dissertationum medicarum, tom. i. Rotterod. 1768), the hydatid growths were supposed to be enlargements of the lymphatic vessels, while their mode of origin was variously explained ; but this inquirer, having paid great attention to the constitution of the cysticercus, and having recognised the relations of the tænia with the hydatids, forthwith gave to the latter the name of Tænia hydatigena, thus establishing their parasitic nature. The observations of Pallas were made on the hydatids which were discovered in the livers of sheep and oxen. They were amply confirmed by Goeze[2] in 1782. Bremser, professor in Vienna in 1821, was the earliest to describe the human echinococcus. This he did by noticing in a hydatid cyst, of the size of a hen's egg, which had been removed by Kern from the sub-clavicular region in a female, thirty other hydatids, " the first of which exhibited to him the echinococci still living."[3] Very important discoveries in relation to hydatids have been made still more recently. The generally received opinion now is, that the echincoccus is just a stage in the development of a tapeworm.[4]

Hydatid tumours are more common in the liver than in any other organ of the body. They consist of a sac, which is closely lined by a thin membranous cyst, and filled with a watery fluid. In the hydatid tumours which are found in man there are usually, indeed almost invariably, seen in the contained liquid a number, varying greatly in amount, of globular bladders or cysts similar to that lining the sac. These small cysts are of very different sizes, some being as small as a pea, while others exceed a walnut in size. It is to these bladders that the name of *Acephalocysts* (*a priv.*, κεφαλή the head, κύστις a bladder) was given by Laennec.[5] In the majority of instances a single hydatid is developed in the liver, but this is not always the case, as two, three, and even more have been found. Sometimes with the presence of a single hydatid tumour in the liver there is associated a hydatid tumour of the right lung in its lower lobe, or sometimes both lungs are thus affected. More frequently still,

[1] Traité des Entozoaires: Paris, 1860. For a full account of the literature of the whole subject the reader is referred to this work of Davaine. Frerichs renders the historical *résumé* of Davaine only scant justice when he remarks, "Eine ziemlich vollständige Zusammenstellung des vorhandenen klinischen Materials lieferte C. Davaine in seinem vortrefflichen Traité."

[2] Versuch einer Naturgeschichte, &c., 1782.   [3] Davaine, p. 353.

[4] The expression *echinococcus* (ἐχῖνος the hedgehog, κόκκος a grain or berry) was applied by Rudolphi to the entozoa of the hydatid cyst, from the cylinder of hooks, or hooklets, surrounding the head.

[5] Frerichs describes the acephalocysts of Laënnec as hydatids containing no scolices ("welche keine Scoleces enthalten"), adding that the existence of such was for a long time doubted. More recently Küchenmeister has designated them sterile echinococci, and they have been considered as an earlier stage of development of the same entozoon by Van Beneden, Davaine, and Lassigne.

hydatid disease of the liver and the spleen coexist. Cruveilheir and Andral have recorded instances of this nature. In addition to these combinations, hydatid tumours of the liver have been found associated with similar disease affecting the mesentery, meso-colon, or omentum, likewise existing beneath the peritoneum, and occupying to a greater or less degree, sometimes so as nearly to fill up, the abdominal cavity. Cases 19 and 20 in Dr. Murchison's work[1] are instances of this nature, and others of a similar character are recorded by different writers.

It has not been satisfactorily ascertained how the development of the echinococci in different situations in the same person occurs. It has been suggested, 1st, that they owe their origin to the absorption of embryos at different periods of time, and, 2nd, that the germs of the more recent cysts which are generated by the older hydatids are carried by the blood to other organs. Dr. Budd, who has discussed this subject at considerable length, and adduced many interesting cases from various writers, adopts the latter explanation, but at the same time is unable to reconcile one great difficulty which exists in the instance of hepatic and splenic hydatids, and thus expresses himself : "The greatest objection to the hypothesis here advanced to account for the tumours in the spleen and mesentery is, perhaps, the improbability that a hydatid germ should pass backwards in the branches of the portal vein, against the current of the blood. It seems more natural to suppose that if the tumours are related as cause and effect, the tumour in the liver is secondary to those of the spleen or mesentery, and not the origin of them. A strong fact against this latter hypothesis is the appearance of greater age in the tumour in the liver in such cases, and the circumstance that while hydatid tumours in the liver alone are not uncommon, it seldom, if indeed ever, happens that hydatid tumours exist alone in the spleen or in the mesentery." [2]

Whether single or multiple the hydatid of the liver consists of an external firm fibrous capsule or cyst, of a whitish or yellowish colour, adhering closely to the surrounding hepatic tissue, and plentifully supplied with branches from the hepatic artery and vena portæ.[3] Within this capsule, and completely filling it, there is a gelatinous translucent grey bladder, composed of numerous concentric hyaline strata. This is styled the mother-bladder of the echinococcus ("die sogenannte Mutterblase des Echinococcus," says Frerichs, whose description we are now following)—that is to say, the embryo which has increased in size to a remarkable extent.[4] Within it is contained a clear watery fluid, with numerous large and small vesicles floating loosely in it, some of which, more particularly the smaller, are adherent

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[1] Clinical Lectures on Diseases of the Liver, page 109.
[2] Diseases of the Liver, p. 443.
[3] This fibrous capsule is usually styled "Balg" by German writers, and "Kyste" by the French. It is the "Folliculus" of Malpighi, Wepfer, and Lancisi, and the "Hydatis externa" of Rudolphi.
[4] Klinik der Leberkrankheiten. Zweiter Band, S. 219.

to the mother-sac. Their size varies from millet or hirse seed to that
of a goose-egg, while in number they may amount to many hundreds
or even thousands. Boudet, Pemberton, Ploucquet, Riverius, and
other writers are cited by Davaine and Frerichs as having recorded
instances in which from five hundred and sixty to nine thousand
hydatids were found in one cyst. The larger bladders sometimes con-
tain smaller ones of a third generation, and in exceptional cases these
in their turn others of a fourth generation. With the number and
size of the daughter-bladders ("Tochterblasen"), and in relation to the
amount of contained fluid, the dimensions of the mother-sac must
necessarily increase; sometimes it reaches and even exceeds the size
of a man's head. Rupture of the mother-sac may take place from
over-distension, and then among the smaller cells only a few shreds
of the former may be discovered. Upon the inner surface of the sac
a number of delicate white particles may on a careful inspection be
noticed; as a rule these are observed in groups, and may be seen
from the outer aspect through the thin walls of the cyst. They are to
be recognised in the fluid also, which is rendered slightly opaque by
their presence. These are the scolices of the Tænia echinococcus in
their various stages of development.[1] The animal is from one-twentieth
to one-sixth of a line long, has a head which resembles that of the
tænia, provided with four suckers ("Saugnäpfen"), and a trunk or
proboscis ("Rüssel"), which is environed by a double crown of hooks, the
number of which according to Küchenmeister ranges from twenty-
eight to thirty-six, or from forty-six to fifty-two. The head of the
worm is separated from its body by a furrow, and presents at its pos-
terior extremity an umbilicated cavity, in which a cord is inserted, and
through its means the attachment of the animal to the inner surface
of the sac is effected. The body presents long stripes passing from the
head backwards and also obliquely from side to side, in addition to
which a greater or less number of rounded calcareous capsules · are
observed. The form of the animal varies greatly, according to the
extension or retraction of the head.

As regards the chemical composition of the hydatid membranes
Frerichs, while affirming that it has not as yet been sufficiently
examined, refers to the proof he afforded in 1848 of their not con-
sisting of some protein compound, as had previously been supposed.
Lücke has rendered it probable that the hydatid membranes contain
chitine,[2] and also that they yield grape-sugar when heated with sulphuric
acid. Davaine speaks of the chemical composition of the hydatid
membranes as being of little practical importance, but he proceeds to
detail at some length that of the contained fluids.[3] The clear liquid

[1] Scolices, from σκόληξ a worm. The term scolex is used to denote a stage in the
development of certain worms, and among these the tænia.

[2] From χιτών a tunic or coat. Chitine was applied by Odier to a peculiar non-
crystallizable organic substance discovered in the wing-sheath of the Cantharis, insoluble
in water and alkalies, soluble in sulphuric and nitric acids, and becoming carbonized
without change in form.

[3] Op. cit. page 371.

from the hydatid cyst does not contain more than a mere trace of albumen; it is charged with a considerable quantity of chloride of sodium, has a density from 1,008 to 1,013, and is either neutral or of slightly alkaline reaction. On the application of heat or addition of nitric acid no coagulation ensues. This is an important fact in relation to hydatid fluids, and may be of service in diagnosis.[1] Heintz and Bordeker have detected succinate of soda in the fluid, but Frerichs, Valentin, Recklinghausen, and Lücke have failed to discover its presence with any certainty.

Hydatid tumours are prone to undergo transformation. They become atheromatous, or they suppurate, and are found to contain either a considerable amount of pus, or of tubercular-looking material: of these the atheromatous degeneration is the really important change. Ruysch is referred to by various writers as having been the earliest to observe the transformation of hydatid growths. " Hydatides," remarked this distinguished professor at Amsterdam in the seventeenth century, "in atheromata, steatomata, et melicerides mutari nulla mihi ambigendi relinquitur ansa: plures enim hoc anno istius modi offendi hydatides, in quibus aliquando materiam pulti, lacti, sero, coagulo, caseoque æmulam reperi." Laënnec, Bremser, Cruveilheir, Charcot, and others have described similar changes. The atheromatous matter discovered in hydatid cysts is principally composed of phosphate of lime, and of an animal matter resembling albumen; it also contains a small quantity of carbonate of lime, of cholesterine, and other fatty matters. Davaine remarks that the presence of cholesterine in atheromatous hydatid cysts is in all probability of general occurrence. Besides the substances already mentioned, there are others less frequently found, and whose presence may be regarded as accidental. These are hæmatoidine and sugar. The former, so far as is yet known, is peculiar to hydatids of the liver.[2]

The position of the hydatid tumour or tumours is not limited to one part of the liver; on the contrary, such are discovered in the right as well as in the left lobe, on the upper surface equally with the under, deep in the substance of the organ, and again projecting from its surface and edges.[3] By the presence of such tumours the form of the liver is variously altered: its size may be greatly increased, so much so, indeed, as to fill the greater part of the abdominal cavity and reach upwards to nearly the right clavicle; but in its earlier stages there may exist a perceptible tumour at one part of the liver, not larger

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[1] Redi was the earliest to point this out. Dodart asserted the correctness of Redi's observations in relation to hydatids in man, and Recamier first recognised this non-coagulability as an important diagnostic feature. (Davaine.)

[2] " Toutes les tumeurs hydatiques dans lesquelles, à notre connaissance, la présence de l'hématoïdine a été constatée, appartenaient au foie." (Davaine.)

[3] "The right lobe of the liver is the ordinary seat of the acephalocysts; the largest are always found at this part." (Rokitansky.) "The most common situation of hydatids of the liver is in its substance, and enclosed in a cyst; but they are occasionally attached to the outer surface of the liver, hanging from it, and occupying more or less of the general cavity of the abdomen." (Matthew Baillie, Morbid Anatomy, 1818, p. 239.)

than an orange—this is the hydatid.[1] By the growth of the hydatid
cyst or cysts, as the case may be, the glandular tissue of the liver
becomes more and more compressed and atrophied.   Those portions of
the organ which are not primarily involved become thickened, and
are said to present at times the characters of a true hypertrophy, the
acini being enlarged and prominent, while no foreign elements are
found.   The larger blood-vessels and bile-ducts are rarely involved in
the hydatid disease; but to this notable exceptions occur, and these
are referred to by Frerichs as follows :—1. The bile-ducts may become
obliterated.   Leroux in one case found no vestige of the hepatic duct,
or cystic, or ductus communis choledochus.   Gadet de Gassicourt and
many others have recorded instances of complete consumption of the
common bile-duct.   2. Communication between the hydatid cyst and
the bile-ducts may be established owing to the destructive pressure
exerted by the former in its progress of growth, just as in the case of
the bronchial tubes, and the intestinal canal, and large blood-vessels a
passage is forced.   Not infrequently the communication of a number
of bile-ducts with the interior of hydatid cysts is found ; and in this
way it is that the contents of the latter become mixed with bile, and
that death of the echinococci is caused.   The presence of bile in the
hydatid cyst would appear to prevent its further growth;[2] and
Frerichs mentions that in most cases of hydatid disease in which he
has observed disintegration and shrinking the sac has contained bile.
Sometimes the hydatid bladders pass from the sac into the open
mouths of the bile-ducts, and becoming impacted there occasion the
dilatation of the ducts, and ultimately pass into the gall-bladder or
the bowel.   By the last-mentioned way the hydatids may be entirely
removed, and the cyst cured.   3. The blood-vessels of the liver, and
especially the hepatic veins, may, in a similar manner to the bile-
ducts, communicate with the echinococcus cysts.

Reference has already been made to the changes which are prone to
occur in hydatid tumours of the liver as their age advances.   Owing
to the increased thickness of the capsule, which at times amounts to
a fibrous or cartilaginous consistence,[3] the development of the echino-
cocci is seriously interfered with ; not only so, but their death ensues
from the impossibility of the obstruction being overcome.   Thus it is
that, as Cruveilhier pointed out, a spontaneous cure is effected.   It is
in the case of hydatid tumours of comparatively small size that the

[1] Murchison, p. 54.

[2] ".The products of inflammation in the matrix, or of the parietes of other cavities
(*e.g.* the pleura), *the bile*, the intestinal secretions, &c., are particularly prone to induce a
maceration and complete solution of the acephalocyst." (Rokitansky.)   Cruveilhier long
ago pointed out that bile appears to be fatal to the life of the parasite.   Its presence
may determine the commencement of a spontaneous cure, or may light up severe
inflammatory action, perhaps terminating fatally.   In reference to this subject we find
Davaine thus expressing himself:—"Nous ajouterons que le contact de la bile a été
considéré dans ces dernières années commes favorable à la guérison du kyste, et que
l'injection de bile de bœuf, pratiquée à plusieurs reprises dans un kyste hydatique du
foie, n'a pas donné lieu à la formation de pus."   (Page 479.)

[3] Frerichs remarks that he has seen a hydatid cyst of the size of a goose-egg com-
pletely surrounded on all sides by a calcareous shell from two to three lines thick.

spontaneously favourable termination or cure is likely to oc
Murchison observes : " Unfortunately, this favourable result i:
for the most part to tumours of so small a size that the
recognised during life.   When the tumour is sufficiently lar;
rise to symptoms and be diagnosed, such an event is so ra
cannot be calculated on."   Where nothing occurs to inter
the growth of the hydatid and the development of the
echinococci, the former may attain a very great size, and r
to exert very injurious pressure not only on the substance of
but on neighbouring organs.   Increasing gradually, it ma
slowly, it finally bursts, and the usual consequence of 1
death more or less sudden.   The directions in which a
tumour of the liver may burst are various.   Some of th
certainly sudden death, others are a little less formidable.
and Frerichs have both fully illustrated this subject.   Dr. N
classifies the direction of perforation under the seven
heads :— 1. Into the right pleural cavity or pulmonary tissu
the pericardium.   3. Into the peritoneum.   4. Through the a
parietes or lower intercostal spaces.   5. Into the stomach or i
6. Into the biliary passages.   7. Into the vena cava inferior.
the rupture into the cavity of the chest is the most freque
occurs almost invariably on the right side. The consequence (
charge of the contents of a hydatid tumour in the liver, tl
opening in the diaphragm, into the pleura, or as in exceptional
into the pericardium, is a very acute attack of pleurisy or peric
the case may be.   When there exist adhesions between th
the right lung and the diaphragm, there is formed a cavity (
or less size in the pulmonary substance, and this cavity n
remain closed, or may ultimately communicate with the
tubes.   In this way the contents of the cyst may be exp(
shreds, if not entire vesicles, are detected in the sputa, togetl
watery fluid containing sugar, and sometimes also bile.   A
thus be effected, but still more likely death through exhau
take place.[1]   Rupture of hydatid tumour of the liver into th(
the peritoneum or into the stomach or intestines is of rarer o
than into the chest.   As in the latter case pleurisy arises,
first-mentioned peritonitis, which being very violent is apt
swiftly fatal.   Rupture in this case may be altogether sp(
or the result of accident—a push, or fall, or strain, or blow.
into the stomach or intestinal canal is by no means so :
rupture into the cavity of the peritoneum.   In the latter c
assuredly takes place in a few hours, but sometimes the a(
rupture is survived for days.[2]   Frerichs saw at Breslau

[1] " A hydatid tumour of the liver may also open into the lung, the hyd;
up, and the patient recover.   Two instances of this have fallen under my c
tion."   (Budd.)
[2] Dr. Budd observes : " It would seem that the bursting of a hydatid i
the sac of the peritoneum causes death as surely, and just as speedily, as th(
an abscess, or as perforation of the stomach or bowel."   (Op. cit. page 436.)

which a hydatid tumour of the liver had been ruptured by a fall: the subject of this case, a young lady, died within a quarter of an hour after the occurrence of the accident. On the other hand, Andral relates the case of a woman, twenty-seven years of age, who was admitted into the hospital La Charité, with all the symptoms of advanced pulmonary phthisis. Suddenly this woman was seized with severe abdominal pain, which was greatly increased by the slightest pressure. During the four days which followed the occurrence of the pain the abdomen, previously pliant and insensible, became swollen, continuing at the same time very painful, the pulse acquired a great frequency, and became more and more feeble, and the greatest pros-tration ensued. Death occurred while the patient was in the act of vomiting green bile. At the post-mortem examination, on raising the liver, a solution of continuity, capable of admitting the extremities of three fingers united, was discovered a little to the right of the gall-bladder: this communicated with a large cavity filled with broken-down hydatids. A renewed examination led to the discovery of the debris of hydatid membranes swimming in the serous fluid within the peritoneum. There were traces of inflammation in the gastro-intestinal mucous membrane. Tubercles at different stages existed in the lungs.[1] The rupture of a hydatid cyst into the stomach or intestinal canal is generally effected through an opening which is narrow and small, and these characteristics it retains, the hydatids being evacuated slowly, and not infrequently at lengthened intervals.[2] Usually the discharge takes place from the bowels, and the hydatids or fragments of them are recognised in the stools; if the stomach has been opened, the hydatids may be vomited;—this, however, is infrequent as compared with the discharge *per anum*. Sometimes the discharge takes place simultaneously in both directions. Again, but this is of rare occurrence, a hydatid tumour of the liver may open externally, through the abdominal parietes, or through the lower inter-costal spaces. Dr. Murchison observes: " Of ten cases of this kind where a spontaneous opening occurred, and of which 1 have collected notes, five terminated fatally." The contents of the liver hydatid may be discharged at or in the vicinity of the umbilicus, or in one of the lower intercostal spaces, and recovery may take place. Frerichs and Davaine refer to an instance of this kind recorded by Felix Plater. It is to be held in remembrance that even after the discharge of the hydatid has taken place through the parietes, with-out causing extreme disturbance to the patient, all risk of dangerous

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[1] Andral, Clinique Médicale. Maladies de l'Abdomen ; xliv. observation.

[2] " L'ouverture de communication qui se fait entre le kyste et l'estomac ou l'intestin est généralement assez étroite, et donne issue aux vésicules avec beaucoup de lenteur : celles-ci sont évacuées au dehors par intervalles plus ou moins éloignés et souvent pendant plusieurs mois." (Davaine.) The much greater danger of the opening into the stomach than into the bowels is well exhibited by the cases which Davaine has collected. Of eleven cases where a hydatid tumour appeared to open into the stomach, six were fatal, while of fifteen in which the opening apparently took place into the intestine only one proved fatal.

consequences is not passed, for suppuration of the cyst may take place, and either fatal peritonitis or exhaustion occur. Those are exceptional cases only in which an opening into the vena cava ascendens occurs. When this accident occurs, the contents of the hydatid cyst, having reached the blood, are carried to the right side of the heart, and thence reach the pulmonary artery, in which, remaining impacted, they give rise to fatal asphyxia. Of this nature is a case related to Frerichs by Professor Luscha. A woman forty-five years of age had for a lengthened period a tumour in the region of the liver, which in no way interfered with her general health. One morning, while stooping in the act of dressing, she became suddenly collapsed, and was in a few minutes a corpse. At the examination of the body after death, the liver near its blunt edge was found to be the seat of an echinococcus cyst as large as a child's head. The sac surrounded the vena cava and was adherent to it. At the lower border of the fossa for the vena cava, the wall of the sac was only a line and a half thick, and here and there was an irregularly indented opening which communicated with the vena cava. Through this opening the bladder had passed to the right side of the heart, and into the pulmonary, the channel of which was completely obstructed.[1]

Another and the last mode of communication of the hydatid tumour in rupturing is with the biliary passages. Dr. Murchison remarks that "it is not uncommon for a communication to be established between a hydatid tumour of the liver and one of the bile-ducts." Davaine, under the head of "Action des Hydatides du Foie sur les Conduits et la Vésicule biliaires," gives several instructive cases of this communication. Case 10 in Dr. Murchison's work is a remarkably interesting one of hydatid tumour bursting into the bile-duct, with discharge of innumerable hydatid membranes *per anum*. In this case recovery took place. Subsequently, however, peritonitis, owing to rupture of old adhesions occurring during the act of vomiting, carried the patient off. Davaine also gives an instance of recovery on the authority of Dr Perrin ("Tumeur dans la Région du Foie: Hydatides et Calculs biliaires rendu par les Selles)." These cases of recovery are certainly remarkable, for although without doubt the biliary canals may become so dilated as to afford a passage to the contents of a hydatid cyst from the liver to the bowel, such cases have almost always proved fatal. Rupture is, however, not the only source of danger in hydatid disease of the liver; there are other and various ways in which death may be induced by their growths. It may result from gradual exhaustion

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[1] Besides the exceedingly interesting case given above, there are other two instances of *hydatid embolism*, or occupation of the vascular system by foreign substances, having their origin in hydatid cysts, recorded at some length by Davaine, and more briefly by Frerichs. One of these occurred in the practice of Piorry, the other in that of Lhonneur. They are placed by Davaine under the head of "Hydatides ou matières d'un kyste hydatique libres dans le cœur et les vaisseaux, et provenant d'un organe étranger au système circulatoire." Of the first of these it is remarked at the inspection of the body :—"On retrouva une substance semblable (la matière puriforme?) dans la veine cave inférieure, dans le cœur droit, dans l'artère pulmonaire et dans ses divisions." (Page 405.)

This is likely to occur when from its great size important organs and functions are interfered with by the tumour, and more especially the respiration.

Again, reference has already been made to the occurrence of changes within the sac, and there are not a few instances in which death has resulted from suppuration or gangrene occurring either within the cyst itself, or external to it. A further source of danger in such circumstances is the establishment of pyæmia, and the formation of secondary purulent depositions. Lastly, secondary hydatid tumours may form in the liver or mesentery, and not only in the adjacent situations, but at a distance from the primary disease.

Case 21 in Dr. Murchison's work is one of secondary hydatid tumour in the spinal canal. Similar instances are on record in which the brain and the heart have been the seats of secondary hydatid tumours.

SYMPTOMATOLOGY.—One of the most notable features in connexion with this subject is the fact that hydatid tumours of the liver may occur and attain very considerable size without their existence ever having been suspected. There is in some instances an entire latency of symptoms.[1] When the tumour is developed deep in the substance of the liver, and when it never attains any considerable dimensions, this is more apt to be the case, for the simple reason that under such circumstances neither pain nor any particular functional derangements are apt to occur. But generally speaking the presence of hydatid growths in the liver is proclaimed by very manifest symptoms. The liver becomes enlarged, and the enlargement continues till a projection more or less marked takes place into the abdominal or the thoracic cavities, in some instances in both directions. This enlargement can readily be determined by palpation and percussion. Unlike the other varieties of painless enlargement of the organ, the fatty and waxy more particularly, the enlargement is not uniform, but usually in one direction only, and thus the form of the liver is greatly altered. On palpation the tumour is free from irregularities of surface, has a smooth globular and elastic feeling, and not uncommonly a distinct sense of fluctuation is recognised. On percussion over the tumour there is felt not infrequently, but by no means invariably, a peculiar vibration or trembling, the so-called *hydatid vibration.* This sign, which was earliest noticed by Briançon and Piorry, is best produced by laying two or three fingers of the left hand gently but firmly over the tumour, and then striking these abruptly with the middle finger of the right hand, or, as Frerichs has observed, when, after percussing, the finger is allowed to rest for a moment on the pleximeter. Pain is not a necessary or other than an accidental symptom of hydatid disease of the liver, and its presence may, generally speaking, be regarded as indicative of inflammation having taken place in the sac. Exceptional instances, however, do occur in which violent pain

[1] " Les hydatides pouvent naître dans le foie, s'y développer, et acquérir un volume considérable sans donner lieu à aucune espèce de symptomes." (Andral.)

is excited by every movement, and by manipulation of the tumour. Frerichs mentions an instance of this nature, in which the diagnosis of cancer was made. On making a puncture, a clear watery fluid escaped, and the pains ceased almost immediately after the tension was removed, and the withdrawal of the fluid permitted the return of the neighbouring parts to their normal position. Jaundice is not a common symptom, neither is ascites or splenic enlargement, nor are indications of disturbed digestion apt to be notable or persistent. With the gradual growth of the tumour, however, there occur difficulty of breathing, a short dry cough, and palpitations of the heart, when the extension is towards the thorax; irritability of the stomach, vomiting, and a confined state of the bowels, when the abdominal organs are subjected to pressure. Œdema of the feet and a varicose state of the veins in the legs, with hæmorrhoidal fulness, occur when the tumour causes pressure on the vena cava. The function of the kidneys is rarely interfered with in hydatid disease of the liver. Instances are, however, on record in which the kidney has become involved through the extension of the disease. Hooklets of the echinococcus have been discovered in the urine, and even portions of the hydatid cyst have been met with.[1] Dr. Murchison relates an interesting case in which the urine contained large quantities of pus, owing to the pressure of a large hydatid tumour of the liver inducing pyelitis.[2] The general health is little if at all interfered with, there is no pyrexia, and only when the enlargement has attained very considerable development, and mechanical obstructions are its necessary result, is there any evidence of impaired nutrition; then and not till then does loss of flesh take place, and is the aspect of cachexia acquired.

This disease, slow in progress, may last many years. A sudden death may occur from rupture, or the same event happening under better auspices as regards the situation of the opening may determine a complete recovery. Frerichs mentions a case which fell under his own observation, in which the disease had existed for at least seven years; and others, the earlier symptoms of which had been noticed two or three years previously. Dr. Budd refers to the case (related in the *Edinburgh Medical and Surgical Journal* for October 1835) of a lady who died at the age of seventy-three. Two hydatid tumours, whose sacs were almost completely osseous, and which contained a thick gelatinous matter and numerous hydatids, were found in the liver. It appeared probable from the symptoms that the tumours had existed from the eighth year of her age.[3]

DIAGNOSIS.—There are certain diseases with which hydatid tumour of the liver may be confounded: these are abscess and cancer of the liver, distension of the gall-bladder, aneurism of the aorta, pressing the

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1 Parkes on Urine, page 213.   2 Op. cit. page 94.
3 Barrier found in twenty-four cases that there were three in which the disease had lasted for at least two years; eight where it had continued from two to four; and four where it had existed from four to six years; while in single instances no fewer than fifteen, eighteen, twenty, or even thirty years, was the period of its continuance.

wards with communicated impulse, pleural effusion on the right
l enlargement of the right kidney from cystic disease. The
of acute symptoms and the tardy growth of the tumour will
distinguish hydatid disease from hepatic abscess. It is well
in remembrance, however, that hydatid tumours of the liver
ect to inflammation and suppuration; and when these have
l, the usual constitutional and local symptoms of abscess are
Dr. Murchison has especially called attention to this possible
of error in diagnosis, and has observed that the recognition of
condition must depend entirely " on the patient's previous
; the fact of a painless tumour having preceded the symptoms
ss, and the absence of exposure to the ordinary causes of tropical
"[1] The absence of smoothness of surface, increased density,
derness, usually distinguish cancer; and as that fatal disease
es, the unmistakeable appearance of cachexia which the patient
s is one never assumed by the sufferer from hydatid disease.
s admits, however, that in the instance of large soft cancers of
er, which offer a feeling of fluctuation on palpation, the diagnosis
no means easy. Jaundice and attacks of colic almost in-
ly precede the distension, with enlargement of the gall-bladder,
may be mistaken for a hydatid tumour. Besides the probable
e of these symptoms in the latter case, there are the absence of
d vibrations and the situation of the tumour, which rarely cor-
ds exactly with that of the gall-bladder, to distinguish the two.
aneurisms are the seat of very notable pulsations—often of
s murmurs—and are generally accompanied by much pain:
circumstances, in addition to the different form of their outline
sist in their recognition. Frerichs has expressed the opinion,
d on personal experience, that hydatids of the liver rising up
e thorax are not unlikely to be mistaken for pleuritic effusion.
of much importance here, to note and accurately to mark the
ter of the upper margin of dulness throughout its entire length.
case of hydatid disease the line is arched upwards, and is
lower level, close to the spine and sternum, than in the
; whereas in pleural effusions the superior line of dulness is
invariably horizontal. Frerichs also observes that the heart
placed further to the left and still more elevated, in the case
datid tumour, than is common in even large pleural effusions.
reat reliance must not, however, be placed on this indication.
doubt still exists, the simple and safe plan long ago suggested
camier may be followed, namely, that of introducing, at the
f most marked fluctuation, a very fine exploring trocar. The
which escapes from a hydatid cyst is distinguished by its clear
y appearance, and, as already mentioned, by the absence of al-
. The fluid when the hydatids are dead may be turbid and
like, but in such circumstances some traces of broken-down
cocci will in all probability be found. Dr. Murchison relates

[1] Op. cit. page 59.

a case where an enormous cystic tumour communicating with the pelvis of the right kidney, and existing for eight years, simulated hydatid tumour of the liver.[1]

Hydatid disease of the liver, when admitting of diagnosis, is to be regarded as a dangerous lesion. The prognosis can never be otherwise than most serious. Owing to the development of the tumour we have observed how contiguous structures and functions are liable to be interfered with and perverted, while the sudden rupture in various directions, which is to be dreaded, is apt to occasion either immediate or rapid death. It is, unfortunately, only in the smaller hydatid tumours—those not appreciable during life—that the favourable termination, owing to spontaneous death of the echinococci, is alone likely to occur.

ETIOLOGICAL CONSIDERATIONS.—The direct cause of hydatid growths in the liver of man is now thoroughly ascertained to depend on the introduction into the stomach and intestines of the ova or embryos of the Tænia echinococcus, which passing into the liver, there undergo development. Of the fact thus stated there can be no longer any doubt; but as to the precise manner in which the transmission is accomplished there is still room for additional inquiry and research.[2] The Tænia echinococcus is a very small tapeworm inhabiting the intestinal canal of the dog, or perhaps more correctly speaking of the genus Canis, including therefore wolves and foxes.[3] This tapeworm, then, not longer than a quarter of an inch, and possessing only four joints, in the last of which, or proglottis, are contained the ova, is an entozoon of the dog chiefly; and its ova, being voided with the fæces of the animal (whether dog or wolf or fox, but not pig as has been erroneously supposed), and becoming mixed with articles which are used for the food and drink of man, are with such carried into the human body. "It is easily understood," remarks Küchenmeister, "how dogs, particularly shepherds' and butchers' dogs, possibly also wolves, and foxes where, as in Iceland, these animals live on sheep, may come at this tænia. In those parts of the country where the breeding of sheep, cattle, and pigs flourishes, the already named animals of the dog race, and especially the shepherds' and butchers' dogs, are readily able to consume the bladders of this

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[1] Case xxiii. op. cit. page 115.

[2] "Envisageant les causes de l'apparition des hydatides à un autre point de vue, on peut se demander pourquoi ces entozaires siégent-ils ordinairement dans les organes abdominaux et thoraciques, fréquence qui chez les moutons et les bœufs est extrèmement prédominante. Il a présenté de ce fait une explication plausible, s'il est vrai que les hydatides doivent leur origine à la transformation ou au développement d'un embrion de ténia. Cet embrion, introduit dans le tube digestif avec les aliments ou les boissons, et ne pouvant vivre ou se développer avant d'avoir subi certaines transformations, quitte cet organe en le perforant, et gagne les parties voisines, soit directement soit par l'intermédiaire des vaisseaux sanguins, lesquels se rendent dans le foie ou dans les poumons." (Davaine, op. cit. page 381.)

[3] Die in und an dem Körper des Leben den Menschen vorkommenden Parasiten, &c. &c. Bearbeitet von Dr. F. Küchenmeister. Erste Abtheilung, S. 150.

species of echinococcus; and we may hazard a conjecture as to the entrance of the eggs and six-hooked embryos into the human body, which can only be similar to that which we have pointed out regarding the origin of the *Cysticercus tenuicollis*, and to which a high degree of probability has been assigned through the experiments of Professor Haubner and myself."[1]

In the sheep as well as in man, the ova of the *Tænia echinococcus* develop hydatids. In the case of these animals the echinococci are liable to be set free during the act of slaughtering, and when the intestines are thrown out as offal they are consumed by dogs, to be in these animals once more developed into tapeworms. Dr. Thudichum observes that the hydatids of man most frequently accompany him to the grave, or are at all events not permitted to continue their dangerous existence; and while man does not add to the multiplication and dissemination of the echinococci, his own liability to the disease is maintained by the cycle of infection which subsists between dogs and sheep.[2]

These considerations have of course a very important bearing on the preventive treatment of hydatid disease of the liver. There are a few other particulars in relation to etiology which call for remark. Hydatids of the liver are chiefly met with about the middle period of life, and are certainly rare both in childhood and in old age. Dr. Budd observes, " Hydatid tumours are most common in persons from the age of twenty to that of forty, but may occur at any age from six years to fifty."[3] The youngest of Frerichs' patients was seven, and the oldest sixty-five years of age; and by far the most of them were in the period of life between thirty and fifty. " Les hydatides existent principalement," remarks M. Davaine, "à l'âge moyen de la vie: c'est de vingt à quarante ans que les cas sont les plus communs. Elles sont presque inconnues chez les petits enfants."[4] Hydatid disease of the liver appears to be of nearly equal frequency in the two sexes. Frerichs and Davaine both notice the statement, resting chiefly on the high authority of Dr. Budd, that sailors enjoy a remarkable immunity from hydatids of the liver. For the rest, we are ignorant of any proclivity to the disease determined by particular occupations and employments. Dr. Budd asserts that " in this country hydatid tumours seem to be more frequent among the poor than among the rich: a circumstance most probably attributable to the fact that the poor dwell in lower and worse-drained houses, and subsist on a diet which contains a much larger proportion of vegetable food."

Different countries appear to be differently affected by hydatid disease. It is endemic in Iceland, where a sixth part of the

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[1] Eodem loco, S. 151.
[2] See "Report on Parasitic Diseases in Quadrupeds used as Food," by Dr. Thudichum. Seventh Annual Report of Medical Officer of Privy Council. London : 1865. Also Murchison, op. cit. p. 72.
[3] Op. cit. p. 448.　　　　　　　　　　[4] Davaine, p. 879.

population suffers.[1]　Frerichs, judging from his own expe
considers the disease more frequent in Breslau and Silesia t
Göttingen, Kiel, and Berlin; while Virchow has found its
prevalence at Wurzburg than in the Prussian capital.　Dr.
points out that the medical writers of India are almost silent :
ing it.　On the authority of Dr. Shattuck, it is very rare
United States.　Leudet says that hydatids are more comn
Rouen than at Paris.　Hydatid disease is certainly very unc
in Edinburgh;[2] much more so than the interesting observat
Bright, Budd, Greenhow, Murchison, and others would lead
believe it to be in London.

A very curious circumstance respecting hydatid growths in
that they have frequently been observed to develop themse
parts of the body which have received some or another form of
Dr. Budd has especially noticed this, remarking, "Of the pu
cases of hydatids of the liver there is a considerable propor
which the tumour seems to have formed soon after a blow on th
and, as was supposed, in consequence of it."　Several of th
recorded by Frerichs had apparently an origin of this kind.

TREATMENT.—This may be briefly considered under the tw
of Preventive and Curative.　A knowledge of the cause of l
disease of the liver is suggestive of its prophylaxis.　To sec
latter, attention to the following rules is required:—1. Dogs sh
prevented feeding on the offal of sheep and other animals i
with hydatids.　Butchers, shepherds who kill for themselve
slaughterers of horses must be careful not to throw such offal
as food.　The duty devolves on the officer of health or physici
community where hydatid disease is liable to occur, of instruct
people, so as to show them, as Küchenmeister has expressed i
unintentional injustice" which may be done in this matter if, a
pointed out, they favour the transmission of the eggs and emb
the tænia.　The same authority suggests that butchers, sla
house men, and shepherds should be instructed to destroy the b
whenever they meet with them, either by burning them or by
them in spirits; and this course, he adds, might be observed
threat of punishment.　Dr. Murchison wisely recommends that
ought to be rigidly excluded from all slaughter-houses or kna

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[1] See "Reflexionen über die Entstehung der in Island endemischen Hydatide
heit, insofern dieselbe durch Echinococcen bedingt ist."　(Küchenmeister,
S. 169.)

[2] "The acephalocyst, even in its usual site, the liver, would appear to be e
uncommon in Edinburgh; as among many thousand dissections which I have ei
formed or seen performed during my connexion with the Royal Infirmary, ther
been a single instance of acephalocystic hydatid or echinococcus either in the li
any other organ, with the exception of this one." (Remarks on a Case of
Tumour in Upper Part of Right Lung, by Dr. Gairdner.　Clinical Medicine, p
　"During six years in which he has acted as Pathologist in the Infirmary, an
other six or eight years over which his observation had extended, he had not p
met with a similar case." (Dr. Haldane, eod. loc. p. 436.)

and dog's-meat ought always to be thoroughly boiled." The same writer suggests that in order to destroy as far as possible the tape-worms generated in the dog, these animals should be periodically physicked, and their excreta buried in the ground or burnt.

The *curative* treatment is either medicinal or operative. Little can be said in favour of the former. It has indeed been supposed that certain medicines are capable of being removed from the blood into the hydatid cysts, and of there killing the echinococci. It was con-ceived by Baumes and others that calomel possessed this power. Laennec prescribed chloride of sodium; Hawkins iodide of potassium.

None of these remedies have ever effected a cure, and it is now well known that iodine does not reach the fluid of the hydatid cyst. Electricity has been employed in Iceland for the purpose of destroying the parasites, and with an apparently successful result. The most important, however, of the means of cure is by *operative* interference; in other words, the evacuation of the fluid contents of the hydatid cyst by means of a fine trocar, and the subsequent closure of the opening. The operation of puncture was practised by Sir Benjamin Brodie, Dr. Bright, and others; while successful as well as unsuccessful cases in their hands resulted. Acute peritonitis and the entrance of air giving rise to suppuration within the cyst, are the dangers to be dreaded, but these have chiefly arisen in those cases where a larger opening than is necessary has been made.

Dr. Murchison and his colleagues in the Middlesex Hospital have satisfactorily shown that these dangers are in great measure avoided by using a very small trocar; and further, that the removal of the liquid from the cyst, which is as thin and limpid as water, is alone sufficient for the purpose of destroying the parent hydatid and its offspring. "The administration of chloroform before the opera-tion," remarks Dr. Murchison, "is not advisable, as the pain is but momentary, and the vomiting sometimes induced by the chloroform interferes with that perfect rest of the parts which ought always to be insisted on for forty-eight hours after the puncture; but if the patient be young or nervous, it may be well to induce local anæsthesia by the ether-spray." The puncture is to be made at the point where the hydatid fluid appears to be nearest the surface, and every care taken to prevent the entrance of air. For the latter purpose it is ad-visable to remove the canula before the whole of the fluid has been withdrawn. When the canula has been removed, the opening should be covered with a piece of lint steeped in collodion, over which a compress and bandage are applied, and for two whole days the patient ought to be kept in the recumbent posture, and at perfect rest. It may be well to give an opiate at once, but its administration must not be delayed if the slightest pain is experienced. Formerly the puncture of a hydatid cyst was considered a very dangerous pro-cedure unless adhesions of a firm character were in existence whereby the escape of the fluid into the pleural or peritoneal cavity was prevented (Frerichs). Now, however, the use of the fine trocar renders

it unnecessary to wait for the formation of adhesions. The walls of the cyst are highly elastic, and the small opening closes immediately after the withdrawal of the instrument. Care should always be taken during the gradual removal of the canula to press the punctured portion of the abdominal wall against the cyst. Dr. Murchison, whose instructive account of the operation we have given, has furnished a table, in which are exhibited the particulars of forty-six cases reported by himself, Dr. Greenhow,[1] and other authors. In thirty-five of these the operation was successful; in ten it was followed by suppuration, necessitating a free opening. Of these ten cases, eight recovered and two died. In one case the patient died from acute peritonitis within twenty-four hours of the operation. When suppuration in the sac has occurred, a large permanent opening is the only justifiable mode of operative interference. The reader is referred to the details of cases furnished by Dr. Murchison and Dr. Greenhow, as affording abundant proof of the propriety of tapping by means of a fine trocar. It appears that the injection of water, solutions of iodine and of iodide of potassium, and bile, practised by various physicians, is quite unnecessary, and at no time free from increased danger.

[1] Pathological Transactions, vol. xviii.

§ II.—DISEASES OF THE DIGESTIVE SYSTEM (*continued*).

G.—DISEASES OF THE PANCREAS.

1. PANCREATITIS.
2. HYPERTROPHY OF PANCREAS, AND OTHER CHANGES.

DISEASES OF THE PANCREAS.

By John Richard Wardell, M.D., F.R.C.P.

General Observations.—The pancreas, like other organs, is subject
to inflammation; it may be acutely, sub-acutely, or chronically
inflamed, and such condition may be primary or consecutive. Pan-
creatitis is most frequently caused by the acute disease or enlarge-
ment of neighbouring viscera. Adhesive inflammation agglutinates it
to surrounding parts; the substance of the gland may pass into the
suppurative state; or it may become indurated, softened, hypertrophied,
or atrophied. Concretions may occur in the ducts and give rise to its
organic disease. Lesion may pervade the whole or only a part of
its substance. It is liable to be metastatically inflamed. Its most
usual morbid condition is that of scirrhus, or carcinoma, and the
head of the organ is the most prone to these deposits. In carci-
nomatous disease of the pylorus and the right lobe of the liver it
sometimes becomes in like manner affected, and passes into chronic
ulceration. In its structure are found fibrous, fatty, tuberculous,
steatomatous, and calcareous materials.

The diseases of the pancreas were, until comparatively recent times,
little understood. Its deep and hidden situation, its proximity to
other organs, and an ignorance of its real functions, were the chief
reasons why its morbid phenomena were less known than the lesions
of other internal parts. The two former of these hindrances to
the study of its pathology can never be overcome, and must needs
prevent the attainment of that more exact diagnosis which can be
arrived at when reviewing the symptoms of the other viscera. The
ancient fathers of physic knew absolutely nothing of its functions or
affections. Hippocrates does not even mention the pancreas, and for
a long series of ages it doubtless received but superficial if any atten-
tion. We must pass on to the sixteenth century before any important
reference is given to this gland. It has been very aptly remarked
that this disregard in the works of the ancients was amply atoned
for by the consideration which was bestowed upon it by the physicians
who flourished two or three centuries ago.[1] Vesalius absurdly imagined
its office to be that of a cushion, to prevent the stomach, when full,
from being injured against the vertebræ. Fernelius ascribed to its

[1] J. J. Bigsby, M.D., Edin. Med. and Surg. Journ. vol. xliv. p. 85. (Edin. 1835.)

disorders diarrhœa, dysentery, atrophy, slow fevers, and other complaints. Schenkius believed it to be implicated in the production of a long catalogue of distempers; Riolanus conceived it to be the seat of hypochondriasis, intermittents, and many other maladies; Sylvius and his followers, amongst whom was Hofmann, connected it with the production of fevers; Highmore said it was the source of apoplexy, palsy, and hysteria; and in the works of Bartholinus, Tulpius, Hildanus, De Graaf, Blancard, Portal, Morgagni, Rahn, and other of the older authors, illustrations of pancreatic disease are often recorded. It was not, however, until Wirsung discovered its proper duct that anything reliable was known of its functions. That was the starting-point of its more accurate physiology, as well as its more correct pathology. Haller classed it with the salivary glands, and succeeding physiologists, amongst whom may be named Magendie, Gmelin, and Lassaigne, concurred in this view, nor have the more recent physiological experimenters dissented from such decision.

A right conception of its structure and office is needful in the study of its pathology. It is a conglomerate gland analogous to the salivary glands, lying transversely across the abdomen, behind the stomach, its greater end or head being surrounded by the curve of the duodenum, and its lesser end extending to the spleen. It is composed of pinkish yellow polyhedral lobules which ultimately consist of arboriform ramifications of minute ducts. Kölliker[1] calls it a compound racemose gland, the smallest lobules of which are rounded microscopical vesicles that possess a proper membrane, and a tesselated epithelium whose cells are remarkable for fat globules, and says these vesicles are connected with small excretory ducts, the latter emptying their contents into canals of larger calibre, and the canals discharging their fluid into the duct of Wirsung which opens into the duodenum.

According to one of the more recent analytical examinations of pure human pancreatic secretion, and which was made by Turner,[2] it is of an orange colour, of marked viscid consistency, and its specific gravity 1·0105. Leuret and Lassaigne pointed out its close resemblance to the saliva, with the exception that it does not possess sulpho-cyanogen. Turner confirms this fact.

With respect to the office which this gland subserves in the economy, it was not until lately that precise conclusions were arrived at. It is true that Eberle many years ago demonstrated that its secretion had the remarkable capability of fluidifying chyme loaded with peptones, and that Pappenheim and Purkinje maintained for it the possession of a distinct digestive power on protein substances; but it remained for Claude Bernard to show one of its most characteristic properties—the change which it exerts upon oleaginous matters subjected to its influence. His investigations went to prove that the pancreatic fluid emulsifies fat, and then converts it into glycerine and fatty acid. That it has also the property

[1] Manual of Human Histology. Translated by G. Busk and T. Huxley. Syd. Soc. vol. ii. p. 137.　　　　[2] Trans. Royal Society of Edinburgh, 1860.

of exciting the transformation of starch into dextrine and grape sugar has been long known. Frerichs avers that the emulsification of fatty food is the result of the joint action of bile and pancreatic juice. Harley thus delivers himself on this point: "There is this important difference between the action of these two secretions on fat, however, that while the bile merely emulsions and saponifies that portion of our food which enters the duodenum in the form of fatty acids, pancreatic juice, on the other hand, possesses the power not only of emulsioning and saponifying fatty acids, but also the neutral fats; indeed, its power seems chiefly to be exerted in the latter." Pancreatic juice is not the only agent which acts upon fat, the bile and intestinal secretions have a similar capability, though in much less degree, and perhaps the conversion is more complete when all these agents act conjointly. Bidder, Schmidt, Frerichs, and Turner believe it to have no influence on albumen. The most recent physiological experiments go to prove that the pancreatic fluid affects the digestion of albuminous substances, and this view is taken by Kuhne,[1] Diakonow,[2] Fudakowski,[3] Schiverin,[4] and Senator.[5] The endeavour has also been made to show that saponification exerts a greater influence in the organism than assisting in the absorption of fatty matters. Radziejewski maintains that soaps can be absorbed and again converted to fat in the body.[6]

This gland may, from a variety of causes, like other glands, differ in its functions, but our means of defining such differences are extremely limited. The intimate vascular and nervous connexion which subsists between it and the neighbouring viscera cannot do otherwise than give rise from time to time to morbid sympathies, and thus, in no trivial manner, influence the great processes of digestion and assimilation.[7] The fluid may be generated in excess, in deficient quantity, or it may be almost or wholly wanting. From a parity of reasoning, founded on a pathological knowledge of other glandular structures, increase of vascular action, and more or less of hyperæmia, are doubtless the conditions coetaneous with and accompanying this augmented flow of the juice. There are instances of its having been given off in very large amounts, and when such is the case, if the lacteals cannot absorb the excess, it must needs then be regurgitated into the stomach, and ejected by an inverted action of the œsophagus, or pass away by the bowels. When secreted in morbid abundance, there are reasons for the supposition that it is then more irritating than normal, and if such be the case the gastric glands, as well as the intestinal surface, would be stimulated to undue action and increase of secretion. The affection known as pyrosis, or gastrorrhœa, has by some pathologists been referred not to the lining coat of the stomach alone, according to the theory once entertained,

[1] Archiv für Path. Anat. u. Physiol. xxxix. p. 130.
[2] Hoppe Seyler's Med. Chem. Unters., Heft ii. p. 241.
[3] Centralblatt, 1867, No. 35.   [4] Dissert. Berlin, 1867.
[5] Virchow's Archiv, xliii. p. 358.   [6] Ibid., xliii. p. 268.
[7] Copland's Med. Dict., art. "Pancreas."

was regarded as the cause. Wedekind long ago believed t
excess of pancreatic juice gave rise to diarrhœa and dysen
rapeutic effects would sometimes seem to render such c
wholly chimerical, because we know that cholagogue pur
capable of producing that kind of discharge. This conditic
sive secretion may be merely functional and transient, o
of organic lesion, and long continued. It is possible that t
may be capable of taking on vicarious action, because we
bowels are prone to do so in renal dropsy, and that this co
tendency is one of the distinguishing qualities of secern
The secretion may be diminished in quantity by the dege
the pancreatic substance into fat, or by its displacement by
deposits; also from mechanical causes, as when tumours of t
liver, or other parts press upon the duct, or when it is blo
a calculus, or its outlet is obstructed by duodenal disea
such is the case, the fæces are apt to be rendered dry and
Brunner extirpated the gland, and then observed the cont
alimentary canal to be deprived of their ordinary mois
copious discharge of fatty matters from the bowels,
will be referred to hereafter, has long been attributed to
or impairment of the pancreatic juice. In some cases of
of the organ, its secretion has been abolished. Bernard s
very slight inflammation of the viscus renders its fluid r
unequal to its proper influence on the chyme; it is then, h
viscid, coagulates by heat and acids more imperfectly, and
power in the emulsification of fat.

GENERAL ETIOLOGY.—With regard to the causes of the
the pancreas, all that can be said must rather be of an
than positive character, as the etiological facts are, when
reviewed, but meagre and inconclusive. That its affecti
tension of morbid conditions of neighbouring parts

predisponent, and thus it is accounted by every authority of note. The long employment of cinchona bark has been supposed to be productive of similar results; such supposition, however, has not been founded on any reliable data. Rahn considered the predisposing cause a pituitous strumous diathesis affecting this in common with other glands. Gout and rheumatism have been blamed, and the obstruction to the menstrual discharge has also been put under the same accusation. Continued dyspepsia and chronic liver affections ought also to be enumerated. Metastasis from the salivary glands and the testicles has been adduced; and from the accounts of Portal, Mondière, and Andral there are grounds for such supposition. A few cases are on record in which the pancreas was found diseased in infants, but the statistics of Claessen show that more than 50 per cent. of the cases occur between the ages of twenty-five and sixty.

GENERAL SYMPTOMATOLOGY.—The physical signs of diseases of this organ are obscure. Palpation affords but vague and uncertain evidence, except perhaps in those cases occurring in thin and emaciated subjects, in whom the organ is malignantly enlarged, and forms an abdominal tumour. In health the viscus can very rarely be detected. It does not move by the act of respiration. Sir William Jenner says: "The healthy pancreas can now and then be just felt in very thin persons with small lax muscles, whose lower dorsal and lumbar vertebræ are somewhat curved forward. I may have satisfied myself that I have felt it half-a-dozen times in my life. The pancreas crosses the aorta and the spine; and when perceptible to touch, it is felt on deeply depressing the abdominal walls about a hand's-breadth below the umbilicus, by then rolling the subjacent parts under the hand (the stomach and colon must both be empty)."[1] This physician also says that in such thin persons it may be taken for malignancy of the transverse arch of the colon, or for aneurism of the abdominal aorta. No symptoms are pathognomonic of pancreatic disease; an assemblage of symptoms indicates the probability of its lesion.[2] Pemberton placed more reliance upon negative than positive evidence, because he could not recognise any set of phenomena which were invariably present. Bright objected to this mode of reasoning, because he conceived it to presuppose an exactitude of knowledge, such as pertains to the other organs, which we do not possess. Siebert of Jena advocates the method of exclusion, a principle which would be erroneous if entirely relied upon; yet its partial adoption constitutes a great aid in leading to a right conclusion. The cardinal symptoms are, a dull, heavy, aching pain deep down below the centre of the epigastric region, which radiates through to the back, left shoulder, and left lumbar space, simulating the pain experienced in renal calculus, and is little increased by pressure; sickness and vomiting, sometimes thirst; constipation, which is occasionally alternated with diarrhœa; languor,

[1] Brit. Med. Journal, Jan. 16, 1869, p. 42.
[2] Diseases of Abdominal Viscera, 3rd Ed., 1814, p. 64.

experiences an increase of suffering after meals, and when th
bitus is on the back. Coughing, deep inspiration, and bend
body forwards, augment the pain. Sometimes the pain re
colic, by coming on in paroxysms, and being mitigated by the
flatulence. The vomited fluid bears a close resemblance to sal
is sometimes, as before observed, ejected in very large qu
Frank and Trumpes state that they have known several pounds
off during the twenty-four hours. Abercrombie says that
twenty-seven cases whose histories he examined, eight had f
vomiting and thirteen had not this symptom. In thirty-nin
selected by Claessen, thirty-five had vomiting. The bowels a
fined in the majority of instances. The diarrhœa may be estin
occurring in one-third of the patients, and the dejections are
stringy, viscid mucus.

When the organ is acutely inflamed the secretion is d
suppressed, because, as Craigie observes, we know it to be t
of inflammation of glandular tissue, that its office is then sus
and it is most likely during the declension of acute sympto
the juice begins to be largely secreted. A secondary train of
mena are exhibited by the progress of the malady, and th
mainly determined by the amount of tumefaction which the
assumes, and the degree of pressure upon neighbouring organs
it exerts. If it press upon the ductus communis, as it fre
does, jaundice, saffron urine, dulness of the right hypochor
congested or absolutely inflamed liver, accompanied by pyrex
ascites, may result; if upon the pyloric end of the stomach, v
becomes urgent; and if upon the abdominal aorta, palpation di
pulsation, and auscultation a bruit similar to that of aneuris
about one-fourth of the cases the enlarged organ can be d
Pressure then *does* give pain. A hard, round, deep-seated l
felt between the scrobiculus cordis and the umbilicus. The di
being interfered with, the assimilative functions are affected;
anæmia, and sometimes this hydræmic state of the blood is re
more manifest by the want of normal tonicity of the blood-
by arterial throbbing in the head and neck, and a humming
ears. The brain remains clear to the last.

One of the most remarkable circumstances connected with

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[1] Med.-Chir. Review, No, xlix.

of the pancreas is the occasional *discharge of large quantities of fatty matter from the bowels.* In 1832, Dr. Bright, in an able paper read before the Medical and Chirurgical Society, brought this subject before the profession, and he thus speaks of the peculiarity in question. "The symptom to which I refer," he observes, "is a peculiar condition of the *alvine evacuation*, a portion, more or less considerable, assuming the character of an *oily* substance, resembling fat, which either passes separately from the bowels or soon divides itself from the general mass, and lies upon the surface, sometimes forming a thick crust, particularly about the edges of the vessel; if the fæces are of a semi-fluid consistence, sometimes floating like globules of tallow which have been melted and become cold, and sometimes assuming the form of a thin fatty pellicle over the whole, or over the fluid parts, in which the more solid figured fæces are deposited." [1] Immediately afterwards, Dr. Elliotson and Mr. Lloyd published similar cases, and the former gave a learned history of the affection, proving that this symptom had been observed by certain of the older physicians more than two centuries ago. Amongst the few instances more recently recorded may be named a very apt illustration by Mr. Clark, of Twickenham. [2]

According to the present state of our knowledge on this particular subject, it may be said that this symptom is dependent upon the absence or a vitiated condition of the pancreatic fluid and deficiency of bile; and physiological experiments and pathological observation confirm this assertion, but the ultimate mode in which the conversion is effected is not so clearly understood. It must be held in remembrance that the distinguishing attribute of the pancreatic juice is to emulsionize fatty matters, but, as I before remarked, other animal fluids possess the same property, though in far less degree. In Dr. Bright's cases two conditions were noticed—malignant disease of the pancreas and ulceration of the duodenum; and that eminent authority connected this product very intimately, if not absolutely, with scirrhus disease. Scirrhus has in repetition been associated with fatty discharge, but it is *not* essential to its formation. In Mr. Lloyd's case there was no scirrhus. In that given by Mr. Clark there was no trace of cancerous disease, and the duodenum was quite healthy, but the proper duct was plugged up with calculi, the organ entirely converted into fat, and, as a consequence, none of the pancreatic fluid could be generated. As previously remarked, the intestinal secretions (especially those of the duodenum), the pancreatic fluid, and the bile are the conjoint factors which emulsionize and saponify the fatty ingesta, but the pancreatic secretion is by far the most important agent in this office, and the disease of this gland explains the voidance of fat in the dejections.

[1] Med.-Chir. Trans. 1832.　　　　[2] Lancet, Aug. 1851.

PANCREATITIS.

Inflammation of the pancreas is an uncommon disease
Meckel, and Andral do not even mention its morbid a
and the last-named declares that it has not been proved
scopy. Such assertion is, however, too absolute, as doub
like other glands, does sometimes pass into the inflamm:
dition. Wedekind, Daniel, and Portal speak confidentl
point; and the examples given by Lawrence, Crampton,
pfeffer, Casper, Gendrin, and lately by Riboli,[1] prove
have been wrong. Its functions not being so importar
economy as the functions of certain of the other viscera,
termination of its inflammation must be still more rare;
morbid state has but in few instances been verified, and the
which pathologists have given of its morbid anatomy ha
entirely consisted of descriptions of the results and compli
inflammation, or of those organic or malignant changes wl
insidiously supervene.

The prominent signs of acute Pancreatitis are deep-seated,
in the epigastrium; distension, sickness, and the vomiting of
greenish viscid fluid; thirst, faintness, moist tongue, constip:
slight pyrexia. In the few illustrations which medical
presents, enlargement could not be detected, partly on acco
distension, and partly because enlargement belongs to th
not acute condition. The characteristics revealed by aut
been redness, brownish redness, or whitish yellowness o
augmented density, increased dryness and elasticity of the
which was in one instance dotted with bloody puncta, and
injection with infiltration of the interlobular cellular tissue,
the lobules more distinct. According to Morgagni, when tl
mation is intense and continued, the organ becomes softer a
more easily than in health. Gendrin saw its proper duct ol
and in Crampton's case the head of the gland was much
Lawrence thus describes what he observed: "The pan
throughout of a deep, dull red colour, which contrasted ver
ably with the bloodless condition of other parts. It was fi
feel externally, and when an incision was made into it, th
lobules felt particularly firm and crisp." Klob is of opi
the inflammation commences in the interlobular cellul
Craigie[2] remarks that the redness and vascularity may be
of transudation after death; but adds, when these are acc
by plastic effusion, pus, induration, or softening, there must l
inflammation. Rokitansky,[3] who regards this gland as bei
to the same morbid changes as those affecting the saliva:
says, there is first tumefaction, then interstitial infiltration,

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[1] Med.-Chir. Rev. No. xlix.
[2] Elements of Path. Anatomy, 2nd Ed. p. 832.
[3] Syd. Soc., vol. ii. p. 178.

disease progresses a sarcomatous condensation of the cellular tissue, consequent upon plastic exudation into the areolae; and that the congestion and reddening attack the acini, which appear to be confounded with the former, and the entire gland is enlarged and indurated. The effusion of coagulable lymph upon its surface may be productive of false membranes by which it becomes attached to one or other of the adjoining viscera, and sometimes the bands of union are dense and organized.

Depositions of purulent matter have been far more frequently seen than the primary conditions of inflammation, and various authors have recorded these collections, amongst whom may be named Tulpius, Bartholinus, Portal, Lieutaud, Gendrin, Percival, and Baillie. They more frequently occupy a portion than the whole of the gland. It has sometimes appeared as if the entire substance of the organ had been abolished and the capsule rendered a membranous bag filled with fluid. In other instances the pancreatic tissue has been found unchanged, its lobules being divided and floating as it were in pus. The suppurative alteration commences in the interlobular cellular tissue in small purulent deposits either in a portion or the whole of the organ, and these punctiform abscesses increase in size and number and ultimately coalesce. Rokitansky says the cellular tissue is then infiltrated with yellow pus, and the acini at a later period become fused. According to Gendrin, the glandular granules are very soft, of a reddish-grey colour, rendered smaller in size, although the whole organ is enlarged. The capsule is thick and inflamed, and when the suppuration is completed the matter is mostly collected in one cavity. It is occasionally mixed with pancreatic juice, which is then a clear yellowish fluid, containing some small curds. The matter is generally inodorous and creamy, but is sometimes greyish white or of greenish hue, of faint, mawkish smell, and is in some instances extremely fœtid. Portal saw two pounds of pus issue from one sac. It is always the result of inflammation. Its collection may be such as to produce injurious pressure on adjacent organs, when ulcerative absorption causes the matter to escape. Gendrin knew it emptied into the duodenum, Gautier into the stomach, and Haygarth into the intestines. It may be extravasated into the cavity of the abdomen, when death speedily ensues. The gall-ducts may be obliterated by pressure, as recorded by Percival and Haygarth, and the proper duct of the viscus may thus be destroyed.

Abscess is liable to occur in the pancreas from metastatic suppurative inflammation. It has repeatedly followed disease of the testicles. Portal cites a case in the instance of a man who had died after castration, where the spermatic cord wasted, and on autopsy a quantity of pus was found in the cord itself, and a considerable abscess in the pancreas. Antonine Petit adduces similar examples substantiating his opinion against the use of the ligature in extirpation of the testes. Tonnellie twice observed pancreatic abscess in puerperal peritonitis. Craigie has noticed it in the bodies of those who have died of ague,

continued fever, and after the suppression of some habitual evacuation, as diarrhœa, hæmorrhoids, and the catamenia. There are no specific symptoms indicative of pancreatic suppuration. Rigors alternated with flushing, in conjunction with the indications enumerated above, might point to such inference.

The metastatic form of inflammation may quickly pass on to *ulceration.* Again, the ulcerative process may be produced by pressure upon adjoining organs, as when the gland is enlarged in malignant disease, and thus a fistulous communication may be established.

The older authors often speak of *gangrene* of the internal organs, as if it were not an uncommon pathological appearance. Bonetus, Becourt, and Greisel give instances of what they conceived to be such condition of this viscus ; and Portal very strangely asserts that gangrene of the pancreas is a frequent result ! Such statements are loose and incorrect, and modern morbid anatomy proves the absurdity of the assertions which were formerly advanced. Gangrene of the pancreas is exceedingly rare. Portal says he beheld the viscus of violet-red colour throughout, its substance being reduced to a pulp, and when opened a black fœtid fluid escaped. Gendrin gives an example which occurred after chronic inflammation.

Storck records an instance of *hæmorrhage* into the pancreas which occurred in a woman aged twenty-eight, in whom violent vomiting came on during the catamenial period, which discharge soon ceased. Dyspnœa, palpitation, faintings, and cold extremities followed. In the course of a month pulsation was felt at the epigastrium, costiveness and want of sleep became urgent, and vomiting, diarrhœa, and wasting ushered in the fatal issue. The pancreas weighed thirteen pounds, and was found filled with coagulated blood. Another case of which I took a note in my reading, but from what author I do not remember, happened in the person of a soldier in one of the military hospitals at Cadiz during the Peninsular war. He laboured under obscure abdominal disease which nothing relieved. The duodenum was found adherent to a reddish-brown tumour, which proved to be the pancreas. Not a vestige of its glandular structure remained. The capsule was a large sac the size of a child's head. It contained coagulated blood and grumous cerebral-like matter. Travers saw the gland ruptured by an accident.

HYPERTROPHY OF PANCREAS, AND OTHER CHANGES.

When there is *hypertrophy* of the viscus, such change has resulted from chronic inflammation, which may have been a long-continued and an insidious disease. An albuminous lymph is deposited in the interlobular cellular tissue, which at length becomes by the absorption of the watery parts condensed and solidified, and may present the appearance of opaque septa. The acini are not much altered, and the organ is rendered larger, and harder and drier. In some cases the hypertrophy is partial. It commonly presses upon the ductus com-

munis, and there is jaundice;[1] Holscher knew it so compress the duodenum as to cause fatal ileus. Riolan relates that he saw it as large as the liver; Tissot beheld it three times its normal size; Rahn found it to weigh four, Westenberg six, and Storck thirteen pounds. Portal, Bedingfield, and others relate accounts of its greatly enlarged volume.

It is sometimes *atrophied*. In cachexia, dyscrasia, and the general debility consequent upon old age, when the vital functions are depressed as in melancholia, and in cases where there are depraved digestion and mal-assimilation, it is sometimes discovered diminished. It also undergoes this change by the pressure exerted by diseased adjacent parts, as of the stomach, liver, spleen, and right kidney, and in aneurism of the aorta. Its arteries have been found ossified, and its bulk reduced; and when the cœliac and mesenteric arteries have been of contracted calibre from osteo-steatomatous deposit, it has been preternaturally small. Its consistency in this state is by no means uniform, it having been seen soft, hard, and of coriaceous tenacity.

It is occasionally *indurated* without being associated with malignancy. The glandular lobules are then the seat of this hardness, and it may accompany hypertrophy or atrophy. It may present a cartilaginous appearance. When thus observed, its colour has been reddish grey; and when cut, its substance has been drier and less vascular than normal. In simple induration the hardness pervades the entire gland, and not a part of it, as in malignant disease. A number of authors have doubtless confounded this condition with schirrus, amongst whom may be mentioned Morgagni, Haller, Tissot, Storck, Baader, and Portal. Modern morbid anatomists define this change with far greater accuracy, yet it is incontestable that its hardened state often passes into absolute scirrhus. It is the most frequently met with in middle life and the aged. Scholler, however, once witnessed it in the infant. *Softening* has been observed in scurvy, scrofula, malignant and eruptive fevers, and in dyscrasia. Such alteration of the organ's consistency is the effect of acute or chronic inflammation. Its softening is mostly associated with increase of size, it is readily lacerable, and is of grey or yellowish green colour.

Fatty degeneration of the pancreas is sometimes met with in accompaniment with fatty decay of the heart, liver, and kidneys, and, according to Rokitansky, this appearance has mostly been found in confirmed drunkards. Fearnside records an instance in which the whole gland was in a great measure converted into fat, and he points out how it differed from mere deposition in the interlobular cellular tissue. Cruveilhier remarks upon the difference between transformation of the glandular substance and fat interposed in the laminous texture. The microscope exhibits the partial conversion of the lobules into and their utter displacement by fat. The entire organ may be transmuted into one fatty mass.

[1] Bedingfield, Todd, Percival, Crampton, and others give such illustrations.

I believe *Scirrhus* to be the most common malady affecting th
pancreas. It may be located in one part or pervade it entirely. I
the majority of examples it is the duodenal end which is diseased
and the gland generally becomes enlarged, but in exceptional case
there is diminution of bulk. I also believe this to be very rarely
primary formation, for other organs nearly always, when carefull
examined, exhibit the same heterologous product. When th
affection has for some time existed, adhesion to adjacent structure
ensues, and there may be open ulceration. By pressure of th
tumour ulcerative absorption takes place, and thus may eventuat
perforation of the diaphragm, erosion of the vertebræ, and ruptur
of the vena cava. This kind of enlargement has been known t
constrict the abdominal aorta and simulate aneurism. The duo
denum by its proximity is more frequently implicated than
any other part; its mucous membrane becomes ulcerated, and
there is adhesive inflammation. The hepatic and common duct
become contracted, sometimes they are blocked up and absolutely
disorganized, and thus the functions of the liver are interfered with
and icterus comes on. The natural secretion of the gland is dimi
nished in quantity, or so changed in quality as to irritate the linin
membrane of the upper part of the alimentary canal; again, th
cancerous deposit sometimes fills up or destroys the duct, when n
pancreatic juice can pass into the bowel, the characteristics of th
chyle become changed, and the fæces are rendered dry. Dr. Owe
Roberts, of St. Asaph, has kindly given me the particulars of tw
cases in which the organ was thus affected, and which recently can
under his notice. One was in a woman of sixty-five, who for lon
had pain in the back, and who died considerably emaciated. Th
pancreas was found large and hard at the duodenal end, and th
common bile duct was obstructed, which accounted for the jaundic
and absence of bile in the stools. The other case was in that of
medical man, whose pancreas was thus diseased, but in him there ha
not been pain in the back nor any jaundice.

The *encephaloid* form of cancer is far less frequently found than th
kind above described, and when it has been noticed there has general
been also medullary sarcoma in some other organ. It is observed
soft, curdy, or cerebral-like matter. In two cases given by Abe
crombie, the encephaloid deposition was arranged in yellow an
white layers. The consequent open ulceration may give rise
fatal hæmorrhage, as related by Videl.

Da Costa has professed to diagnose cancer of the pancreas from i
other maladies, but the truth is we have no just rules of observan
to point out any real distinctions. Pain at the epigastrium, sicknes
vomiting, and constipation have been instanced as cardinal sympton
by Claessen and others, but they have also been given as the sympton
of simple inflammation of the gland, therefore they cannot be regard
as pathognomonic of carcinoma.

Bright once saw *Fungus Hæmatodes* of the pancreas, and the sa

statement has been made by other authors, but it is extremely rare. There are specimens of *Melanosis* of the organ in the museums of the Royal College of Surgeons and St. Bartholomew's Hospital. *Tubercle* has been noticed by a few pathologists, but only in phthisical and strumous subjects, and as the accompaniment of the same condition in other organs. *Steatoma* is another product which some writers have mentioned, but I believe it to have been but another name for tubercle. As in the salivary glands, *calculous concretions* are formed in the pancreas. They are mostly discovered in the main duct, but they have been also observed in its minute ramifications, presenting the appearance of small particles of white earthy matter. They are of irregular surface, and vary greatly in size and number. They may not be larger than a millet-seed or equal to a hazel-nut, and nearly one hundred have been counted. Schupmann [1] found one measuring one inch and six lines in length, and which weighed more than three drachms. Wollaston and Fourcroy proved them to consist of phosphate of lime in combination with some animal matter. *Cysts* have been spoken of, but dilatations of the ducts have been mistaken for such growths. Cysts in this organ may have their origin in areolar expansion or from distension of the duct; the same occurs in ranula with obstructed submaxillary duct, and a like cystiform dilatation is sometimes seen in the Fallopian tube. Professor Turner, of Edinburgh, saw an excellent illustration of cystiform dilatation of the pancreas from obstruction of the duct, in the body of a man who died with cancer of the head of the viscus. The pancreas at first sight looked like a multilocular cyst; but examination showed the cyst-like dilatations to be expanded acini filled with the thickened secretion of the gland. [2]

DIAGNOSIS OF DISEASES OF PANCREAS GENERALLY.—It is only approximately that the diagnosis can be accomplished. The functional affections of the pancreas cannot be recognised, and it is only when its maladies have made progress, and the other viscera have become implicated, that they can with anything like certainty be inferred. The low degree of sensibility with which the organ itself is endowed, and the great sensibility of organs with which it lies in juxtaposition, its depth in the abdomen, the inconsiderable effect which its lesion exerts on the circulatory, nervous, and secretional systems, and the resemblances which the diseases of the liver, stomach, and duodenum bear to those of this gland, are some of the many causes constituting this difficulty. Strict regard should be paid to the symptoms before enumerated, and the complaints incident to the stomach, liver, spleen, and duodenum should as far as possible be excluded. Kreysig and Hohnbaum place most confidence in the sickness and the slimy fluid which is vomited. Wasting, and the discovery of particles of fat in the fæces, are by Lussanna considered the most reliable signs. But we now know that fat may be passed in the alvine evacuations when the

[1] Hufeland's Journal, April 1841.
[2] Paget's Lectures on Surgical Pathology. Edited by Turner. London, 1863, p. 393.

duodenum and not the pancreas is diseased : hence this symptom is not pathognomonic. Again, with regard to the pain : Abercrombie says there may be great disease with little or no pain. Swelling of the parotid glands has been noticed as a vicarious affection. Cancer of the pylorus, enlargement of the liver, spleen, mesenteric and mesocolic glands, encephaloid tumours, impaction of fæces in the transverse colon, should, if possible, be excluded. Da Costa commends the exclusive method, and doubtless the attempt to diagnosticate will be facilitated by the observance of such means in addition to a due regard to those conditions which are looked upon as the more positive indications.

TREATMENT.—From what has now been said relative to the diffi-culty which there is in diagnosticating disease of the pancreas, the treatment must needs be more doubtful and less satisfactory than the treatment addressed to organs whose morbid conditions can with greater certainty be interpreted. There are no medicines which have a special power in counteracting its maladies. The treatment must be conducted on those general and acknowledged principles which would be equally applicable to other internal parts. If we believe acute inflammation to be present, the ordinary antiphlogistic measures should be adopted. Cupping or leeches may be used; and if there be hot skin, quick pulse, and great pain, and the patient is robust and strong, moderate general blood-letting may be employed. Emollient cataplasms, hot and repeated fomentations, terebinthinate and opiate epithems often give relief. When the more acute symptoms have declined, blistering, tartar-emetic ointment, croton-oil liniment, belladonna and opiate plasters, or the linimentum hydrargyri, or the emplastrum hydrar-gyri, can be tried. If the pancreatic affection be considered metastatic of parotitis, a blister should be applied to the parotid gland. The bowels should be kept open by laxatives, such as castor-oil, the con-fection of senna, or the compound rhubarb pill, with a little of the extract of henbane. The empirical plan of some of the older phy-sicians, of giving an opiate at bed-time and a gentle aperient in the morning, may be followed. If sickness and vomiting be urgent, hydrocyanic acid, and small doses of morphia in a mixture of mucilage or almond emulsion, are likely to be serviceable ; effervescing draughts and creosote in pills, and small quantities of brandy in iced water, may be given. If there should be much pain, opiates in some form must be had recourse to ; and if the irritability of the stomach be such as to re-ject them, they should be administered hypodermically or by enemata. If we are impressed with the conviction that the affection is carcino-matous, palliatives can alone be used with advantage, and the surest of these is opium. Mondière, in such cases, gives importance to revulsives, and praises the use of the moxa. The diet should be bland and nutritious, such as nourishing soups, jellies, milk, and farinaceous food. If there be acidity in the stomach, milk and lime-water should be given. Rest in the horizontal posture should always be insisted upon.

§ III. DISEASES OF THE RESPIRATORY SYSTEM.

A. DISEASES OF THE LARYNX.

§ I. PRIMARY DISEASES OF THE LARYNX.
    ACUTE LARYNGITIS.
    CHRONIC LARYNGITIS.
    MORBID GROWTHS.
    NEUROSES.

      DISEASES OF THE MOTOR SYSTEM.
        PARALYTIC AFFECTIONS.
        SPASMODIC AFFECTIONS, LARYNGISMUS STRIDULUS.
      DISEASES OF THE SENSORY SYSTEM.

§ II. SECONDARY DISEASES OF THE LARYNX.
    IN ACUTE AFFECTIONS ; THE EXANTHEMATA.
    IN CHRONIC AFFECTIONS.
      LARYNGEAL PHTHISIS.
      SYPHILITIC DISEASE OF LARYNX.

APPENDIX, ON THE USE OF THE LARYNGOSCOPE.

DISEASES OF THE LARYNX.

BY MORELL MACKENZIE, M.D.

IN order to facilitate the treatment of this subject, Diseases of the Larynx have been divided into PRIMARY and SECONDARY. The first includes all those conditions in which the larynx is the part first affected, and where the disease is generally, though not necessarily, of a purely local character. The second embraces those conditions where the laryngeal affection is a complication of a previously developed (acute or chronic) morbid state of the system. The primary affections are the inflammations, the morbid growths, and neuroses; the secondary are the occasional phenomena met with in the acute exanthemata, in phthisis and in syphilis. The classification is based on convenience. The limits allotted to this article forbid my occupying space by defending the arrangement, or by anticipating and explaining away any possible charge of apparent inconsistency in carrying it out.

SECTION I.

PRIMARY DISEASES OF THE LARYNX.

ACUTE LARYNGITIS.

DEFINITION.—Inflammation of the lining membrane of the larynx, in which the vessels of the submucous areolar tissue may or may not participate, characterised by dysphonia, or aphonia, dyspnœa, and stridulous breathing, cough, slight pain in the larynx—generally referred to the *pomum Adami* and increased on pressure externally—and dysphagia. There is generally high constitutional fever.

SYNONYMS.—*Latin*—Cynanche Laryngea, Angina Laryngea, Angina Epiglottidea; *French*—Laryngite, Catarrhe Laryngien; *German*—Katarrlische Kehlkopfenzündung; *English*—Inflammation of the Larynx. Laryngitis is by some subdivided into Mucous Laryngitis (the *Laryngite muqueuse* of the French), and Sub-mucous, or Œdematous Laryngitis (*Laryngite œdemateuse*).

CAUSES.—(a) *Predisposing.*—That relaxing habits predispose to th
disease is rendered probable by the fact, that residents in towns ar
more liable to it than those living in the country (Niemeyer); and c
the former, those engaged in indoor occupations are much mor
susceptible than those much exposed to the weather. At the Hospita
for Diseases of the Throat, laryngitis is much more often met wit
among tailors, shoemakers, porters, and people thus engaged, tha
among coachmen, cab-drivers, policemen, and others who are con
stantly exposed to the most inclement weather. Previous inflamma
tion, and of course repeated previous attacks, render the part parti
cularly prone to be affected. Males are more liable to it than female
and adults than children; but it proves far more fatal to the young
more than four-fifths of the mortality occurring before the tenth yea

(b) *Exciting Causes.*—Cold draughts of air, whether inspired or bea
ing on the neck externally, exposure of the body in general to col
and especially allowing the feet to remain wet and cold for any lengt
of time, are circumstances which in some people may give rise to th
disease. Violent functional efforts (in giving the word of comman
preaching, singing, &c.), and straining the parts in coughing, ar
not uncommon causes of it. Dusty air and irritating vapour
ought, perhaps, to be considered as the *traumatic* causes; the
are both probably sometimes concerned in the production of th
disease, without even the patient being aware of their operation
The catarrhal form of the disease is often propagated from th
nares, and œdematous inflammation sometimes from the pharynx
Extension of the disease occasionally takes place from below, th
bronchial tubes being first affected; but the opposite sequence mor
often takes place, the laryngeal disease passing off with the occurrenc
of bronchitis.

SYMPTOMS.—The approach of the disease is generally insidious, ar
a slight catarrh may suddenly become a most serious affection.

(a) *Subjective Symptoms.*—The patient complains at first of a slig
dryness or soreness of the throat, or he may have nothing more than
feeling of roughness, or a tickling sensation with disposition to coug
or there may be a sense of constriction about the throat, and slig
difficulty of swallowing; but the period at which this symptom sup
venes, as well as its degree, depends on the part of the larynx first a
most affected; in other words, it occurs at an early period, and
greatest when the epiglottis or ary-epiglottic folds are mu
affected, and later, and to a less degree, when the more internal pa
of the larynx are attacked.

In severe cases all the true laryngeal symptoms become great
aggravated. There is often a sensation as if a foreign body we
lodged at the part, the breathing becomes extremely embarrassed, ar
the patient feels great anxiety about getting his breath. In fatal case
the restless agony of impending suffocation generally gives way at la
to a comatose state.

(b) *Objective Symptoms.*—(1) *Vocal.*—There is generally dysphonia in the early, aphonia in the later stages. The cough is at first clear and shrill, then harsh and croupy, finally aphonic. It is generally frequent, and often paroxysmal. Its exact character and variations, however, depend on the particular part of the larynx which is affected.

(2) *Respiratory.*—The inspiration is at first a little prolonged and wheezing, afterwards very much lengthened and accompanied with stridor. In the later stages there is a kind of groan in expiration. In addition to these sounds mucous râles can generally be heard on auscultation over the larynx. As the calibre of the larynx becomes contracted from œdematous infiltration and spasmodic approximation of the vocal cords, the patient expends all his energies on the respiratory process. Sitting up in bed he desperately clutches the bed-clothes, and, in his violent efforts to get breath, the shoulders are seen to rise and the whole chest to heave.

(3) *Laryngoscopic Signs.*—In the early stages, and in mild cases, the mucous membrane is merely seen to be of a bright red colour; the hyperæmia is, as a rule, diffused, though sometimes there may be distinct injection of the vessels. In severe cases œdema soon appears, the parts affected being seen to be red, swollen, and semi-transparent. When the epiglottis is acutely inflamed, it frequently presents the appearance of a raised ridge in the median line, with two large tumours on each side ; the valve is in fact folded upon itself, and only its upper surface is visible. This condition occludes the view of the larynx. When the ary-epiglottic folds are attacked, their shape becomes very irregular ; the ventricular bands are sometimes seen to be in a highly turgid state, and in this case they eclipse the vocal cords. If the latter are visible, they are of a bright red colour, slightly swollen, especially posteriorly ; their sharp free edge is rounded, their mobility is impaired, and on inspiration their normal action is occasionally seen to be reversed, the glottis tending to become closed instead of open.

(4) *Miscellaneous Symptoms.*—The laryngeal secretion is generally very scanty, tenacious, and difficult to expectorate; in favourable cases, where the disease is passing off, it may become thick, purulent, and abundant. In the early stages, though not generally till a few hours after the local symptoms have manifested themselves, there are signs of inflammatory fever; the tongue is white and furred, the tip and edges being generally red. The pulse is frequent and hard, the skin hot, and the face flushed. At a later stage the constitutional conditions resemble that of hectic, the skin under the immense respiratory efforts being bathed in perspiration, and the pulse small, feeble, frequent, and irregular. The countenance is of an ashy pallor, the lips purple, and the eyeballs protrude from the dark halo which surrounds them.

Course and Termination.—The acute stage seldom lasts more than three or four days, and I have seen a case terminate fatally in twenty-four hours. Death has been known to occur in seven hours.[1] It is

[1] Dr. Wood. Pract. Med. vol. i. p. 780.

rare for the symptoms to remain serious after the fifth day, unless kind of chronic œdema sets in. The disease may terminate in any the following ways :—(1) Spontaneous resolution may occur. (2) Re solution may be brought about by therapeutics. (3) The acute symp toms may pass away, and chronic congestion remain. (4) Death ma take place very suddenly, from the combined effects of œdematou swelling and spasm of the glottis, less suddenly from the former caus acting alone, or slowly, and often preceded by delirium from the effect of exhaustion and imperfectly aërated blood. (5) Threatened suffoca tion may be averted by the operation of tracheotomy.

DIAGNOSIS.—In very young children it is impossible to distinguis between acute laryngitis and croup ; but, where the laryngoscope ca be used, the presence or absence of false membrane can of course b ascertained at once. Even with this instrument, however, the essentia nature of the morbid process cannot always at any early period b ascertained, as the apparently simple inflammation may be an earl stage of the plastic form of disease.[1]

Laryngismus stridulus differs by its very sudden accession, by i generally occurring during sleep, by its passing off and leaving th child in an apparently normal condition as regards the larynge symptoms and respiration, and by the absence of constitutional fev Spasm of the glottis in adults is easily differentiated by the gener symptoms, and still more so by the employment of the laryngoscope

PATHOLOGY.—The disease is essentially a simple inflammation of t mucous membrane, and submucous areolar tissue of the larynx ; t danger of the disease being in proportion to the extent that the la named structure participates in the morbid process. When the deep tissues are affected, the products of inflammation accumulate benea the lining membrane and cause the tumefaction which in this situatic is attended with such imminent risk. When the inflammatory proce is superficial, its effects are of less importance. The character of t secretion becomes altered, being at first clear and gummy in characte and afterwards containing an increased quantity of pus corpuscle There is partial destruction and imperfect formation of the norm epithelial structure, but the process scarcely ever leads to ulceratio The danger is not due to the œdematous swelling alone, but al to the spasm of the glottis which the infiltration causes—partly b reflex action, partly by direct irritation of the adductor muscles the vocal cords.

MORBID ANATOMY.—The superficial appearances, a few hours aft death, resemble those described under the head of Laryngoscopic Sign In children, the mucous membrane is generally slightly softened, an of a bright red colour. In adults, on the other hand, the rednes seldom remains after death, as in those cases which prove fatal th

[1] See article "Croup," Vol. I. p. 258.

activity of the morbid process is in the submucous tissue. The product of the inflammatory process is generally of a serous character, but it may be sero-purulent, or may even be of the nature of what is called "healthy pus." In the latter case, the condition is that of diffused abscess: circumscribed abscess—as far as I am aware—never occurs as a sequel of acute inflammation of the larynx. The effusion, however, is much more frequently of the serous character. It generally collects in those parts where the areolar tissue is most lax : thus the epiglottis and the ary-epiglottic folds are the parts which are both the most frequently distended, and which become the most swollen; next to them the ventricular bands (false vocal cords) are most commonly affected ; the vocal cords may be a little tumefied, but they are rarely swollen to any extent. The muscles are often saturated with the serous fluid. If the patient survives the acute stage and dies from other causes, the parts previously swollen and œdematous, present a peculiarly sodden and shrunken appearance.

PROGNOSIS.—In giving an opinion as to the danger, the age of the patient is the most important consideration. In early life, that is, before the development of the larynx has taken place at puberty, the disease is always attended with great danger. As regards adults also, a very serious opinion must always be given. The danger depends on the amount of œdema present; and though tracheotomy remains as a last resource, there is always a risk of the disease extending down the windpipe, so that both bronchitis and pneumonia often supervene.

THERAPEUTICS.—If the case come under observation at a very early period, it is really quite impossible to tell whether the disease is a simple catarrh of the larynx, or is likely to turn out a violent inflammatory affection. Under these circumstances a system of rational expectancy must be adopted ; a warm, moist, and uniform temperature enforced, and gentle diaphoretic and mild purgative medicine administered.

The same kind of treatment cannot be carried out in the case of children as where the patients are *adults*. The following will be found useful for the latter class of patients :—In the early stage, and in slight cases, an inhalation of hot steam, or steam impregnated with the volatile principles of benzoin, or hop or conium, may be used.

The following forms will be found serviceable :—

     1. ℞ Tinct. benzoin. comp. fl. dr. j ad fl. dr. ij :
to be added to a pint of water at 150° F., and inhaled (from a quart jug with a narrow neck, or from a special inhaling apparatus) for ten minutes every three or four hours.

     2. ℞ Ol. lupuli ℳ xv.
          Mag. carb. lev. gr. x.
          Aquæ ad fl. oz. iij. M.

A teaspoonful in a pint of water at 150° F., and used as No. 1—for

five to eight minutes, three times a day. The addition of a scruple of camphor to three ounces of any of the foregoing will be found useful, if a rather more stimulating effect is desired. The juice of conium in the following form is often beneficial :—

3. ℞ Succi conii fl. dr. ij.
Sodæ carb. gr. xx.

Mix and add to a pint of water at 150° F., and use as No. 1.   Or—

℞ Coniæ gr. ⅛ to gr. ¼.
Sp. vini rect. fl. dr. j.

Mix and add to a pint of water at 150° F., and use as above. Where there is much pain, or tendency to spasm, chloroform (ten to thirty drops) may be added once or twice at intervals of five minutes during the inhalation. These remedies can be used alone or in combination. If crescent inflammation of the pharynx accompanies the laryngeal hyperæmia, the local action of guaiacum administered in the form of lozenges will often prove most beneficial.

If, however, the disease makes head under this treatment, and the parts are acutely inflamed, *without being œdematous*, an attempt may be made to restrain the crescent inflammation by the application of a strong solution of nitrate of silver (60 gr. ad 1 fl. oz.), or perchloride of iron (120 gr. ad 1 fl. oz.) ; or chloride of zinc (30 gr. ad 1 fl. oz.) ; or chloride of aluminium (gr. 60 ad 1 fl. oz.). Solutions of nitrate of silver, still largely employed by the profession, have not proved more serviceable in my hands than the mineral astringents, whilst they more often cause spasm and nausea. Inhalations of atomized liquids may be tried, and among these tannin (5 gr. ad 1 fl. oz.), ferri perchloridi (3 gr. ad 1 fl. oz.) are most likely to do good. If the inflammatory process is not arrested by the action of these remedies, the œdema, which is almost sure to supervene, should be treated by free scarification with the aid of the laryngoscope, and a properly constructed laryngeal lancet. Should this treatment, however, be impossible or ineffectual, and should the dyspnœa be of a threatening character, tracheotomy must not be delayed. In these cases the result of the operation is especially favourable. General blood-letting, leeching, blistering, mercury, and antimony, were the most weighty remedies of the profession twenty years ago ; but they cannot be put in the balance against the topical treatment which the laryngoscope renders possible. Non-depressant emetics, however, such as sulphate of zinc (20 gr. to 30 gr.), sulphate of copper (5 gr. to 10 gr.), in plenty of warm water, are sometimes useful where there is much œdema ; and leeching and blistering may be conveniently resorted to by country practitioners who have not the opportunity of applying remedies with the aid of laryngoscopy.

Treatment of Children.—As it is impossible to distinguish between infantile laryngitis and croup, the treatment must in effect be the same for each disease.[1] In addition, however, to the treatment recom-

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[1] See article "Croup," Vol. I. p. 261.

mended by Mr. Squire, scarification, as described below, may sometimes be performed with the greatest advantage.

VARIETIES.—Acute inflammation has been here described in its most complete and severe form; but it can easily be understood that congestion of the larynx, or *Sub-acute Laryngitis*, may come on very suddenly, remain for a few days, and pass away without any further development. The hoarseness which often accompanies faucial catarrh is due to this cause, the vocal cords being in this case the part of the larynx most affected. The symptoms either give way, or the disease assumes the character of chronic laryngitis.

Traumatic Laryngitis may, perhaps, be considered as belonging to the province of surgery; but it is desirable briefly to call attention to that form which occurs to children from swallowing boiling liquids. Children of the poorer class are often allowed to drink tea from the spout of the teapot, and when left alone they attempt the same feat at the boiling kettle. Instant inflammation of the pharynx and orifice of the larynx sets in, and in two or three hours, or even sooner, the epiglottis becomes greatly swollen and œdematous.

Scarification, first recommended by Lisfranc,[1] and since by Busk,[2] Tudor, and others, is the most rational treatment. The age of the patient generally renders the use of the laryngeal mirror out of the question; but the fauces should be illuminated as in laryngoscopy. In children, under these circumstances, the swollen and œdematous epiglottis can be seen in an erect posture at the back of the tongue. It may be scarified with a gum lancet, or a curved, sharp-pointed bistoury, which should be quite blunt (or covered with strips of plaster) up to within two or three lines of its extremity. Emetics either before or after scarification are often useful. The pressure which the act of retching exercises on the œdematous tissue, perhaps proves beneficial in consequence of the mucous membrane rupturing, and allowing the aqueous matter to escape. A strong solution of nitrate of silver or of some other mineral astringent may sometimes arrest the crescent inflammation *before œdema has taken place*. The local abstraction of blood is recommended by some, and Dr. Bevan[3] has reported four severe cases successfully treated by application of leeches to the margin of the sternum, an emetic followed by a cathartic, two grains of calomel every half-hour, and mercurial inunction. Scarification, however, fairly and fully carried out, ought to supersede all other treatment.

Tracheotomy, from which *à priori* the most satisfactory results might be anticipated, is not a very successful operation in these scalded throats;[4] but nevertheless recourse must be had to it when other remedies fail, and the dyspnœa threatens death.

Journal Général, Année 1825.   [2] Lancet, August 13, 1859.
Dub. Quart. Journ. of Med., Feb. 1860.
See Med. Times and Gaz. vol. xix. p. 366; and Brit. Med. Journ. Jan. 14, 1860.

CHRONIC LARYNGITIS.

DEFINITION.—Chronic inflammation of the lining membrane of the larynx characterised by hoarseness or loss of voice and generally by more or less cough.

SYNONYMS.—*Latin*—Laryngitis chronica; *French*—Laryngite chronique; *German*—Der chronische Katarrh der Kehlkopfschleimhaut. For other synonyms see "Laryngeal Phthisis," which disease was formerly confused with chronic laryngitis.

Causes.—The causes of the disease are the same as those indicated under the head of Acute Laryngitis, to which disease it often proves the sequel. The chronic forms of inflammation, however, more frequently extend from the pharynx, and the effects of continuity of texture are often seen in chronic alcoholism and the abuse of tobacco. It is also more frequently caused by functional excesses.

The great and sudden development of the larynx which takes place at puberty in males, is often attended by a mild form of laryngitis— the so-called "cracked voice" of boys being always associated with marked congestion of the vocal cords. There seems also to be a rare constitutional condition, where there is a tendency to chronic inflammation of many of the mucous canals. Four such cases have come under my notice; the patients were all men over fifty years of age. I have at present a gentleman under my care suffering from chronic laryngitis, slight thickening of the walls of the lower third of the œsophagus, gastro-intestinal derangement, and chronic cystitis. The influence of age and sex is very marked, adult males being by far the most common sufferers, and children the rarest.

SYMPTOMS.—*Subjective.*—The patient's sensations are not generally very vivid, a tickling feeling being generally all that is complained of; in some cases, however, a pricking or burning pain is felt. The congestion of the vessels and perhaps the presence of an altered secretion causes in some cases a frequent desire and effort to clear the throat.

Objective.—(1) *Vocal.*—Impairment of function is the most characteristic symptom of the disease. It varies in degree from slight modification in tone, to complete loss of voice. It is characteristic also of this form of hoarseness *in the early stage*, that it is most marked when there has been rest of function for some time. Thus a person with slight chronic congestion may be extremely hoarse on attempting to speak after being silent for some time, but the voice may become almost normal after the function has been exercised for a few minutes. The improvement probably depends on the quickened capillary circulation, and stimulated nerve-force of the part. It has its analogy elsewhere. In dysphonia, dependent on feeble approximation of the vocal cords on the other hand, the voice is strongest when

first exercised, and gradually becomes weaker as it continues to be exercised. Sometimes the voice is clear and natural in its ordinary tones, and the discordance is only observed when powerful exertions are made (as in singing, acting, public speaking, &c). The cough is generally rather frequent, but it may amount to nothing more than a hawking or "hemming" noise, and sometimes it is almost altogether absent. On the other hand, it may be the most troublesome symptom.

(2) *Respiratory.*—The respiration is not materially affected, though moist râles can usually be heard over the larynx.

(3) *Laryngoscopic Signs.*—The congested condition of the lining membrane of the larynx is at once apparent on using the laryngoscope. The hyperæmia may be general or partial. The following is the order of frequency in which the mucous membrane over the different parts is affected :—First, the capitula Santorini; secondly, the ventricular bands; thirdly, the epiglottis; fourthly, the vocal cords, and least frequently the ary-epiglottic folds. The redness generally fades off gradually into the healthy coloured membrane, but injection of the minute vessels is sometimes apparent on the epiglottis and vocal cords. On the former the injection is generally arborescent, on the latter the arrangement of the vessels is usually linear, along the attached side of the vocal cord. Sometimes one vocal cord is seen to be bright red whilst the other is of its usual white colour, and the congestion may even be limited to a small portion of a cord. Sometimes the anterior half or third of the cord, sometimes the posterior portion is affected ; or even a section of the whole length of a cord may be injected, whilst the rest remains of a normal colour. In the latter case it is always the outer attached portion of the cord which is congested. Small pellets of mucus are often seen sticking to different parts of the laryngeal membrane ; and in cases of long-standing disease, the larynx has the appearance of being very much dilated and covered with secretion ; on the other hand, the membrane may look dry and glistening. It is often noticeable that on attempted phonation the vocal cords do not thoroughly approximate, the congestion of the membrane interfering with the action of the muscles.

Miscellaneous Symptoms.—The varying character of the expectoration may be inferred from what has been already stated, but it may be observed that it is seldom abundant, unless the laryngeal affection is complicated with bronchitis. The constitution does not generally suffer, but there is occasionally some sympathetic irritation.

Course and Termination.—The tendency of the disease when once fully established is to remain stationary, or the symptoms may disappear for a short time, and then recur. The disease, in old people, is always complicated with chronic bronchitis, and the symptoms of the later affection mask and outweigh in importance the morbid phenomena dependent on the chronic laryngeal disease. The principal danger is from chronic œdema coming on, but this is an exceedingly rare complication. In some cases, especially between the ages of twenty and forty, persistent chronic laryngitis appears to predispose to the

development of phthisis, but it is difficult to tell how far the laryng
hyperæmia is concerned as a cause, and how far as a consequence.

DIAGNOSIS.—An accurate opinion can only be formed by a car
laryngoscopic examination. It is of the first importance to obse
whether there be thickening or not; and in the former case to no
carefully whether there be merely inflammatory tumefaction, œde
tous infiltration, or tuberculous exudation.

In œdema the swelling is generally of a bright colour, and ha
characteristic transparent appearance; in phthisis, on the other ha
the thickened parts are generally of a dull colour—though the sur
may be congested, and the swelling generally presents the appeara
of a solid tumour (see " Laryngeal Phthisis "). In all cases of chro
laryngitis of some months' standing, the lungs must be most caref
examined, the history of the patient and that of his family clo
investigated, and his general condition inquired into, before a deci
opinion as to the nature of the disease is given.

PATHOLOGY AND MORBID ANATOMY.—The disease is essentiall
chronic inflammation of the lining membrane of the larynx, in wh
the vessels of the areolar tissue participate very little. Enlargem
and tortuosity of the small vessels is found in cases of long-stand
congestion, and occasionally, but very rarely, dilatation of the laryng
canal takes place.

PROGNOSIS.—The disease never terminates fatally, unless some co
plication arises; on the other hand, it is often difficult to cu
especially in old people.

THERAPEUTICS.—Local remedies are the most important agents
the treatment. These are commonly called " caustics," but their act
seems rather of an astringent character. Any of the following n
be used :—Ferri perchlor. (60 gr.), ferri persulph. (60 gr.), ferri su
(120 gr.), cupri sulph. (10 gr.), zinci chlorid. (30 gr.), zinci a
(5 gr.), zinci sulph. (10 gr.) aluminis (30 gr.), alum. chlor. (60 g
dissolved in an ounce of water or glycerine. The latter veh
through its denser consistence, is better adapted for keeping
a prolonged action on the part. The chloride of zinc solu
is the remedy which I most frequently employ; but provided t
the application is made accurately and sufficiently often, it re
matters very little which solution is used. The application sho
be made daily for the first seven days, every other day the sec
week, twice in the third week, and so on—gradually lengthening
interval between the application. This is a general rule, but it n
be modified according to circumstances. In cases where there is
cessive secretion from the larynx (laryngorrhœa) the local applicat
of turpentine sometimes does good, though these cases are very troul
some to treat. On the other hand, where there is long-standing hy

with diminished secretion—where the mucous membrane looks
nd shining—the remedy which I have found most successful is
lic acid (from half a drachm to a drachm of the pure white
lic acid to an ounce of glycerine). These local remedies can be
pplied with the aid of the laryngoscope—the laryngeal mirror
held in the left hand and a camel's hair brush (fixed to a slender
f aluminium at an angle of about 95° or 100°, and fastened in a
en handle) in the right hand. Those who do not employ the
goscope should hold the patient's tongue well out, in such a posi-
hat the posterior wall of the pharynx can be seen, and should
pass the brush down between the latter and the base of the
e. In this way the remedy is likely to reach the desired desti-
n: the old method of pressing down the tongue with a spatula
sing a flexible sponge probang could only end in failure. Instru-
s of the syringe character are quite unnecessary for the application
medies to the larynx, and they give rise to more irritation than
ple brush. Powdered substances likewise cannot be recom-
ed—they are as a rule either inert or injurious. Great benefit
wever, sometimes derived from inhalation—either of steam im-
ated with some stimulating volatile principle, or of atomized
ds of an astringent character. For the steam inhalations the
wing formulæ will be found useful :—

> ℞ Ol. pini sylvest. fl. dr. ij ad fl. dr. iij.
> Mag. carb. lev. gr. lx to gr. xc.
> Aquæ ad fl. oz. iij. Mix.

aspoonful to be added to a pint of water at 150° F., and inhaled
ve minutes twice or three times daily.

> ℞ Creasote, fl. dr. iij.
> Glycerine, fl. dr. iij.
> Aquæ ad fl. oz. iij. Mix.

aspoonful to a pint of water at 150° F. as above.

> ℞ Ol. juniperi Ang. ♏ xx.
> Mag. carb. lev. gr. x.
> Aquæ ad fl. oz. iij. Mix.

aspoonful for each inhalation as above.

o either of these the addition of a scruple of camphor is often
iceable after the mixture has been used for about a week.
r spray inhalations the following ingredients are most to be
mmended : the proportions given are always for one ounce of
r; and the quantity to be used each time should be from two
drachms to half an ounce of the solution :—

> Alum, 10 to 20 grains.
> Tannin, 1 to 20 grains.
> Perchloride of iron ⅛ to 2 grains.
> Ditto, 2 to 10 grains (in hæmorrhage).
> Sulphate of zinc, 1 to 6 grains.
> Chloride of zinc, 2 to 10 grains.

snia, with diminished secretion—where the mucous membrane looks dry and shining—the remedy which I have found most successful is carbolic acid (from half a drachm to a drachm of the pure white carbolic acid to an ounce of glycerine). These local remedies can be best applied with the aid of the laryngoscope—the laryngeal mirror being held in the left hand and a camel's hair brush (fixed to a slender rod of aluminium at an angle of about 95° or 100°, and fastened in a wooden handle) in the right hand. Those who do not employ the laryngoscope should hold the patient's tongue well out, in such a position that the posterior wall of the pharynx can be seen, and should then pass the brush down between the latter and the base of the tongue. In this way the remedy is likely to reach the desired destination : the old method of pressing down the tongue with a spatula and using a flexible sponge probang could only end in failure. Instruments of the syringe character are quite unnecessary for the application of remedies to the larynx, and they give rise to more irritation than a simple brush. Powdered substances likewise cannot be recommended—they are as a rule either inert or injurious. Great benefit is, however, sometimes derived from inhalation—either of steam impregnated with some stimulating volatile principle, or of atomized liquids of an astringent character. For the steam inhalations the following formulæ will be found useful :—

> ℞ Ol. pini sylvest. fl. dr. ij ad fl. dr. iij.
> . Mag. carb. lev. gr. lx to gr. xc.
> Aquæ ad fl. oz. iij. Mix.

A teaspoonful to be added to a pint of water at 150° F., and inhaled for five minutes twice or three times daily.

> ℞ Creasote, fl. dr. iij.
> Glycerine, fl. dr. iij.
> Aquæ ad fl. oz. iij. Mix.

A teaspoonful to a pint of water at 150° F. as above.

> ℞ Ol. juniperi Ang. ℳxx.
> Mag. carb. lev. gr. x.
> Aquæ ad fl. oz. iij. Mix.

A teaspoonful for each inhalation as above.

To either of these the addition of a scruple of camphor is often serviceable after the mixture has been used for about a week.

For spray inhalations the following ingredients are most to be recommended : the proportions given are always for one ounce of water; and the quantity to be used each time should be from two fluid drachms to half an ounce of the solution :—

> Alum, 10 to 20 grains.
> Tannin, 1 to 20 grains.
> Perchloride of iron ¼ to 2 grains.
> Ditto, 2 to 10 grains (in hæmorrhage).
> Sulphate of zinc, 1 to 6 grains.
> Chloride of zinc, 2 to 10 grains.

It is almost unnecessary to observe that the voice should be exercised as little as possible. For singers, actors, clergymen, and others whose occupations require them to use the voice much, rest of the vocal organ is of the utmost importance. When complete silence cannot be enforced, the least possible exertion should be made in speaking—the patient should, in fact, whisper. All direct sources of irritation must of course be removed. Thus, if the uvula is much elongated, it must be amputated before a radical cure can be effected. As the pharynx is almost invariably more or less affected, astringent lozenges will be found very useful. Tannin, rhatany, and kino may often be prescribed in this form with great advantage.[1] Change of climate is often very beneficial. Generally speaking, a warm dry atmosphere suits best, but provided there is no humidity the temperature is not so important. Thus, in parallel cases, I have seen equal benefit follow from a short residence in Algiers and a few weeks spent at Cromer or Margate. The warm relaxing climate of the south coast is generally injurious; but cold winds, especially those of an easterly character, often give rise to sub-acute inflammation. The waters of Ober-Salzbrunnen and Ems (source, Kränzchen) are especially recommended by Niemeyer, who observes, that "the influence of these waters, so manifestly favourable in many cases, cannot be explained by physiology." Weilbach, Eger, Kissingen, and Marienbad are recommended by other German writers, whilst French physicians praise the waters of the Pyrenees. Where suitable atmospheric conditions cannot be selected, the patient must wear a respirator, when the weather is at all cold and damp, and must protect the neck and body generally by warm and suitable clothing. Medicines and hygienic treatment may be necessary in some cases, and must vary according to circumstances.

Varieties.—(1) Chronic Glandular Laryngitis. (2) Phlebectasis Laryngea.

(1) Chronic glandular laryngitis is a variety of chronic inflammation in which the minute racemose glands are principally affected. The credit of first noticing it is generally given, in this country and in America, to Dr. Horace Green of New York, but according to French writers it was described by Professor Chomel (*Gazette Médicale*, April 1846) at least six months earlier. It has many *synonyms:* thus we have dysphonia clericorum (mal de gorge des ecclésiastiques, clergyman's sore-throat), angine glanduleuse, laryngite granuleuse (ou granduleuse), follicular laryngitis, follicular disease of the pharyngo-laryngeal membrane, and tubercular sore-throat. As the glands of the larynx are all of the racemose variety (Kölliker), the term follicular laryngitis is obviously incorrect, and glandular laryngitis designates the condition more accurately. The causes of the affection are the same as those which give rise to simple inflammation. The French, indeed, consider that it is of an "herpetic" nature, but this term is used in such a

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[1] These lozenges have been prepared for me by Messrs. Bullock and Reynolds, 3, Hanover Street, who will be happy to give the formulæ to any practitioner.

vague way by French authors, that it really has no definite meaning. The morbid process of the larynx often results from an extension of the disease from the pharynx, in which situation *the follicles* are principally concerned; it may, however, originate in the larynx and afterwards reach the pharynx. The disease is not peculiar to the clergy, nor is the chronic laryngitis, from which they often suffer, as a rule, of the glandular character. The disease might with equal truth be called "costermonger's sore-throat." It is often associated with indigestion, but whether there is any causative relation between these conditions is uncertain. The symptoms are the same as those of simple chronic laryngitis, but perhaps milder—weakness of voice, fatigue after speaking, a constant inclination to clear the throat and swallow the saliva or perform an act of deglutition, being the principal morbid phenomena. With the laryngoscope the enlarged orifices of the glands may sometimes be seen on the epiglottis and the posterior part of the vocal cords as pale specks on the congested membrane, or as small red circles on the pale membrane; the other laryngeal appearances do not differ from simple laryngitis, except that the approximative action of the vocal cords is more often feeble and imperfect. Constitutional debility was thought by Dr. Green to be a characteristic phenomenon, but there is very often no general weakness or evidence that the system at large is at all affected. As regards the pathology of the disease, it may be remarked that it is essentially a disease of the secretory system, the normal secretion of the minute racemose glands, instead of being clear and transparent, becoming thick, white, and opaque. The morbid process is essentially mild, but very chronic, in character. The treatment should be the same as that recommended for simple chronic laryngitis. Solutions of the crystals of nitrate of silver (from two to four scruples of the salt to an ounce of distilled water) were strongly recommended by Green and since by other writers, but they do not seem to me to act more beneficially than other mineral astringents. The sulphuretted waters of the Pyrenees, especially of Les Eaux Bonnes, are viewed by the French as almost specific in their action; and several patients that I have sent there have derived undoubted benefit from the use of those waters, where the voice remains weak after the glandular disease has been cured. Benzoic acid lozenges often act very beneficially as nervo-muscular stimulants.

(2) Phlebectasis laryngea,[1] or venous congestion of the larynx, is an extremely rare affection. It may depend on general or local causes; that is to say, it may occur "in persons affected with a morbid preponderance of the venous system" (Hasse), or may be due to a local strain. The symptoms are generally slight; some alteration in the voice, an uneasy sensation in the larynx, and perhaps a more or less frequent cough, being the principal morbid phenomena. The laryngoscopic appearances may be thus described:—In mild cases, when the disease is very limited, extremely fine dark vessels may be seen running along the upper border of the ventricular orifice: in more

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[1] This disease was first described by the author in the Lancet, July 6, 1862.

severe cases there is less regularity in the distribution of the distended veins, which may be observed on the ventricular bands, vocal cords, and arytenoid cartilages. Cases have come under my notice in which streaks of blackened mucus adhering to the larynx have been mistaken for varicose veins. This error needs only to be mentioned to be avoided. This condition of the larynx, independently of the inconvenience it occasions, is probably attended with some danger, as it most likely predisposes to passive œdema. The disease should be treated by the application of strong astringents; and of these a saturated solution of tannin in glycerine is the best Constitutional remedies of a tonic and invigorating character should also be used.

MORBID GROWTHS.

DEFINITION.—New formations, whether of simple or malignant character, appearing as distinct tumours, projecting from the mucous membrane of the larynx, and more or less separated by a line of demarcation from the tissue from which they grow.

SYNONYMS. — *Latin* — Polypus Laryngis ; *French* — Polypes du Larynx ; *German*—Kehlkopfpolypen, Neubildungen im Kehlkopfe ; *English*—Polypus of the Larynx Warty Growths, Warts, New Formations, Excrescences, Cancerous Growths, &c.

NATURAL HISTORY.—*Causes.*—Benign growths in the larynx are probably almost always dependent on local hyperæmia, and therefore their primary causes must be sought for under the head of Laryngitis. Chronic inflammation of persistent character but low degree is, probably, the condition most favourable to their development. In young children the disease is often attributed to an attack of croup, and it probably does originate sometimes in this way. The presence of a warty growth in the larynx, however, produces symptoms closely resembling, and very likely to be mistaken for, those of croup. Neither syphilis nor phthisis are predisponents. The evolution of cancerous growths in the larynx (as elsewhere) is dependent on laws of development which are not understood.

SYMPTOMS.—(*a*) *Subjective.*—The early symptoms are very vague, as the chronic laryngitis which precedes the new formation causes the same sensations (see Chronic Laryngitis). Patients occasionally complain of a feeling of something sticking in the throat, and when the growth is pedunculated there is sometimes the sensation of a body moving about in the larynx. This, however, is quite the exception. Even in cases of true cancer there is seldom much pain. Difficulty of swallowing is generally present, if the growth attains to any size,—

especially if it affects parts concerned in deglutition or projects into the food-tract. Where the growth is large, shortness of breath is experienced.

(b) *Objective Symptoms.*—(1) *Vocal.*—The voice is generally but not necessarily hoarse. Dysphonia is more common than aphonia. Small warts more often destroy the function than the larger and polypoid varieties (Czermak). The voice has sometimes a kind of paroxysmal or intermittent character, being one moment almost normal, and at the next very hoarse, or even quite suppressed.

(2) *Respiratory.*—The breathing is embarrassed if the tumour is large, and there is sometimes stridulous breathing. The extent to which the respiration is affected bears a direct relation to the respective sizes of the growth and the laryngeal canal.

On auscultation, moist sibillant râles may be heard over the larynx, and a valvular murmur has been described as being very characteristic of the presence of a morbid growth (Rühle): it is not, however, to be depended on. The cough varies in character and in frequency in different cases, and sometimes is of a croupy character.

(3) *Laryngoscopic Signs.*—With the laryngoscope, the disease is generally at once revealed. The appearances vary according to the pathological nature of the tumour. *Papillomata*, which are the most common of all benign growths, vary in size from a grain of mustard to a walnut—their most common size being that of a large split-pea. These growths have generally a mammillary, lobulated, or cauliflower configuration. They are generally of a lighter colour than the surrounding mucous membrane, and most frequently grow from the vocal cords. They are generally sessile and multiple. *Fibrous tumours* are seldom seen of such small size as those of papillary character, and they are also much less common. They vary in size from a split-pea to an acorn, and have usually a smooth surface. They are generally single and pedunculated, and in seven of the fourteen cases that have come under my notice, the growth sprang from the vocal cords, and was confined to those parts. *Fibro-cellular tumours* are comparatively rare, and represent only about 5 per cent. of all benign laryngeal growths. They sometimes attain the size of a cherry, but are generally smaller. They are invariably pedunculated, generally sessile, and usually of a pale colour. *Cystic tumours* are seen as round egg-like projections, and as they usually give rise to some local irritation, they are themselves red and surrounded by a hyperæmic area. The other kinds of growth are too rare to require a special description. In cancer there is generally irregular thickening of some parts, and destruction of others. The former process usually precedes the latter. Destructive ulceration is most characteristic of epithelial cancer, and thickening of the encephaloid variety. The epiglottis and ventricular bands (false vocal cords) are the most common seats of true cancer. In encephaloid cancer the parts are often so much displaced that there is difficulty in recognising them. The epiglottis may be pushed completely on one side, and an ary-epiglottic fold or ventricular band

may be so much swollen and inflamed as to cover the parts situated below, on the opposite side of the larynx. Growths, especially small ones, on or about the vocal cords, are apt, as Türck first pointed out, to give rise to functional paralysis of one of the vocal cords.

(4) *Miscellaneous Symptoms.*—Occasionally the presence of a morbid growth is proved by the patient coughing up particles which can be examined microscopically. Sometimes the growth rises out of the larynx, and can be seen when the mouth is widely opened.[1] In children it is sometimes possible to introduce the finger into the larynx and feel the growth ; but the small size and soft structure of these warty productions makes it almost impossible thus to distinguish them. The expectoration is generally increased and altered slightly in small benign growths—very much in the case of larger ones and in cancer. Where the growth is small and benign, constitutional symptoms may be, and generally are, altogether absent ; but where the tumour is so large as to interfere seriously with respiration, the system at large is likely to sympathise : in some of these cases there is irritative fever, whilst in others the constitutional symptoms are more those of hectic. In the case of true cancer, the characteristic cachexia is present.

Course and Termination.—In the case of non-malignant growths, the symptoms generally develop themselves slowly, taking several months for their evolution ; after attaining a certain degree of severity, they often remain stationary, unless some complication, as œdema or spasm of the glottis, occurs. The latter condition is likely to supervene if the growth is large ; and it gives a paroxysmal character to the symptoms. The advanced symptoms are those of impending suffocation. Epithelial cancer usually gives rise to more distressing local symptoms ; there is more expectoration, and deglutition is often difficult and painful. In encephaloid cancer the symptoms progress more rapidly, the fatal termination usually taking place within a few months of its first appearance. This, however, is not always the case.[2] The patient sinks from the combined effects of gradual suffocation, slow starvation, and the intrinsic nature of the disease.

DIAGNOSIS.—Tumours in the larynx cannot well be mistaken for any other disease, if a laryngoscopic inspection can be made. The possibility of eversion of the ventricle must not, however, be forgotten. The tubercles of syphilis are seen as irregular whitish prominences on the congested mucous membrane—the posterior wall of the larynx being their most common site. The thickening of laryngeal phthisis is not so great as that of true cancer, nor has it the defined character of the benign kinds of growth. It is not quite so easy to distinguish between the benign and the malignant epithelial growths. The former, however, are more strictly defined, and never (unless quite accidentally)

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[1] Horace Green on Morbid Growths in the Larynx, p. 62, and Rayer, Maladies de la Peau, tome ii. p. 422.
[2] See specimen No. 28, Series xxv., in the Museum of St. Bartholomew's Hospital.

ulcerated, whilst in the latter there is generally irregular thickening from interstitial exudation, and frequently ulceration. The microscope cannot be relied upon for differential diagnosis should particles be expectorated, or removed during life with the aid of the laryngoscope. Several cases have come under my notice where the histological features were decidedly those of cancer, whilst the clinical history was of a totally opposite character. The laryngoscopic appearances and constitutional symptoms furnish much more important indications in relation to the differential diagnosis of the various kinds of growth than the microscopic examination.

PATHOLOGY.—The non-malignant kinds of tumour are essentially local productions—the result of a perverted nutritive process in which growth is excessive and development imperfect. Hence the formation of tissues of abnormal size and morbid structure. They are probably always associated in their origin with local hyperæmia. In the case of malignant growths, in addition to the local changes, there are constitutional influences in operation which will be found fully described elsewhere.

MORBID ANATOMY.—The benign growths found in the larynx are Papillomata, Fibromata, Fibro-cellular Tumours, Cystic Tumours, Sarcomata, and Lipomata, and they are here enumerated in their order of frequency. The malignant growths belong either to Epithelial or Encephaloid varieties.

Papillomata are by far the most common of laryngeal growths, three-fourths of all the benign cases being of this nature. The papillary growths, "in their general form and arrangement, have many points of resemblance, but on the enlarged scale, to the papillæ, which in various localities constitute natural projections from free surfaces, more especially from the skin and mucous membranes. Their basis substance is formed of connective tissue, which is continuous with that which normally exists in the part; whilst the free surface is covered by an epithelium, which may vary in thickness and its number of layers according to the seat of the tumour. Blood-vessels and even nerves enter into the interior of the papillæ."[1] These papillary growths vary in size from a pin's-head to a cherry, and may even attain a larger size; but after reaching a certain magnitude their growth sometimes ceases spontaneously. They grow rather quickly, especially in their early stages. To the naked eye they have a rough lobular laminated appearance, and they are generally soft and even friable to the touch. Dr. Andrew Clark has kindly made microscopic examinations of many of the growths of this kind, which I have removed during life with the aid of the laryngoscope. He has

[1] Lectures on Surgical Pathology. By James Paget, F.R.S. 3rd Edition, p. 591. For an elaborate description of these growths, the reader is referred to Virchow's Krankheiten Geschwülste, vol. i. p. 334 et seq. This eminent pathologist regards Papillomata as a sub-order of his large division of Fibromata.

described them generally as "consisting of more or less perfect c
nective tissue, clothed with many layers of epithelium." In some
them "enlarged racemose glands, the terminal vesicles of which w
filled with minute nucleated cells and granular matter," were obser
Dr. Andrew Clark thus described one case :—"The growth was fo
to consist of two sets of particles, one membranous, the other wa
or obscurely papilliform. The membranous portions consisted of f
twenty to thirty layers of scaly epithelium, surrounded and penetra
by a confervoid growth. The epithelial cells composing the la
were polygonal, flattened, nucleated, and easily affected by w
alkalies and acids. The nucleus of each cell was oval, abruptly defi
rather large in proportion to the containing cell, in most cases
rounded by a clear halo, and in some showing signs of division.
papillary portion consisted of simple outgrowths of nucleated con
tive tissue, and rudely-formed blood-vessels, clothed with nume
layers of scaly epithelium, similar to those already described. S
of the papillæ exhibited large vacuoles or spaces filled with col
matter, which in one or two instances had burst through the cove
epithelium." Papillomata show a greater disposition to recurre
than other growths.

Fibromata.—The fibrous tumours of the larynx, like those occur
elsewhere, are found to consist of bundles of white fibres diverg
and interlacing in various directions. They seldom attain a la
size than a hazel-nut, and when removed show no disposition
recurrence.

Fibro-cellular Tumours of the larynx are comparatively rare. T
growth is rather slow, but they sometimes attain a huge size. "T
consist," says Mr. Paget, "of delicate fibro-cellular tissue, in
undulating and interlacing bundles of filaments. In the intersti
liquid or half-liquid substance, nucleated cells appear imbedded i
clear or dimly granular substance." Unlike the mucous polypi
the nose, they exhibit no disposition to recurrence.

Cystic Growths are still more uncommon in the larynx. I h
only met with two cases—both situated on the epiglottis. They
however, occasionally found near the ventricular orifice. They c
tain a thick, white, semi-fluid sebaceous material, which on mi
scopic examination is found to consist of epithelial cells undergo
fatty degeneration, with perhaps a small amount of the pro
secretion of the glandulæ. When thoroughly emptied, they sh
no disposition to form again.

The other kinds of benign tumour, such as Lipomata, Vascu
Tumours, Hydatids, &c., are too rare to require a special descript
in an article of this kind.

Cancer.—Under Cancer we must consider the two kinds which
found in the larynx. These are (1) malignant epithelial, and (2) en
phaloid. (1) Malignant epithelial. Though considered with referer

to other morbid conditions of the larynx, epithelial cancer is not common ; as compared with many other situations, the larynx must be regarded as a favourite site. The epiglottis is the part most frequently attacked, and next to it the ary-epiglottic folds. It often gives rise to large ragged ulcerations. The disease may be primary, or consecutive to disease in adjacent parts—the pharynx, œsophagus, or thyroid gland. Cancer in the larynx is seldom secondary, in the sense that the term is generally employed by pathologists. (2) Encephaloid cancer is less common than the epithelial variety, but, like it, is often consecutive. It is characterised by its greater tendency to induce interstitial exudation and consequent thickening, and perhaps by its lesser proneness to ulceration. As already remarked, it often produces considerable displacement.

In addition to the different forms of morbid growth here described, others are said to have occasionally existed in the larynx. Ryland quotes a case of "hydatids" in the ventricle of the larynx, from Andral, which gave rise to the symptoms of a foreign body at that part.[1] The same author speaks of "cartilaginous tumours" in the larynx, and gives several illustrations of supposed tumours of this sort. There is no reason why enchondroma should not be developed in this part, but it is exceedingly rare, and has not been observed by any pathologist of note. Erectile tumours are described as occurring in the larynx, by Rokitansky.

PROGNOSIS.—The prognosis as regards a fatal termination depends on the nature of the growth, and, as regards recovery of function, on whether the growth can be removed. Cases of true cancer, of course, always prove fatal; whilst the other kinds of tumour ought never to do so.

THERAPEUTICS.—Small growths not giving rise to functional disturbance, and showing no disposition to increase in size, need not be interfered with ; but where the neoplasm is large, and where it shows a disposition to grow, it should, if possible, be removed with the aid of the laryngoscope. This removal may be effected by evulsion, for which various instruments are suitable. Most to be recommended are forceps of different lengths, and opening in different directions ; thus, for a growth on the ventricular band, a pair of short forceps opening in the lateral direction is required, whilst for a growth on or below the vocal cords a much longer forceps and opening in the antero-posterior direction are indicated. My tube-forceps are very useful, especially if the larynx is small. *Ecraseurs* are not, as a rule, to be recommended, but Stoerck's *écraseur*, which has a rigid metal loop protecting the wire, is, however, often serviceable. No force should be used in the evulsion of growths. Where they cannot be removed without undue effort, crushing of the growth will often cause atrophy.

[1] Ryland, Diseases f the Larynx, p. 226.

In some cases, the base of the growth may be incised with the laryngeal lancet; or if the tumour be too large for this method, forceps having a cutting edge may be employed with perfect safety and with the best results. Galvano-cautery, on account of the pain it generally causes, and the risk of subsequent inflammation, cannot be recommended. Caustic solutions I have not found to be of any service except in those cases where the growths, small in size and symmetrical in situation, are of the nature of condylomata. In these cases the tumours possess but a very feeble organization, and are often dispersed by the application of caustic or astringent solutions.

Should the growth be very large and threaten suffocation, and should severe spasm be induced by attempts at removal through the upper opening of the larynx, the operation of tracheotomy should be performed; the neoplasm may be afterwards removed, either with the aid of the laryngoscope or by division of the thyroid cartilage. In cancer, relief can sometimes be obtained by the inhalation of simple hot steam, or steam impregnated with various sedative principles, as recommended in the treatment of Acute Laryngitis. I have seen a few cases in which removal of a malignant growth, situated so as to seriously impede respiration and deglutition, has been attended with very great temporary relief. One such case is reported in Pathological Transactions, vol. xxi. p. 53.

NEUROSES (NERVOUS AFFECTIONS OF THE LARYNX).

Under this head are included—(1) Diseases of the Motor System, and (2) Diseases of the Sensory System.

DISEASES OF THE MOTOR SYSTEM.

This division embraces—first, Paralysis of the Muscles of the Vocal Cords; and secondly, Spasm (or Spasmodic Approximation) of the Muscles of the Vocal Cords.

The varieties of paralysis are so numerous, and their nature so different in different cases, that it is better to consider them separately under the following heads:—(1) Paralysis of the Adductors of the Vocal Cords; (2) Paralysis of the Abductors of the Vocal Cords. These may again be divided into (*a*) unilateral and (*b*) bilateral paralysis.

Bilateral Paralysis of the Adductors of the Vocal Cords.

DEFINITION.—A condition in which, owing to the non-approximation of the vocal cords on attempted phonation, there is loss of voice.

SYNONYMS. — *Latin* — Paralysis Glottidis, Aphonia Paralytica, Aphonia; *French*—Aphonie; *German*—Kehlkopflähmung; *English*—Functional Aphonia, Hysterical Aphonia, Aphonia.

CAUSES.—Debility and hysteria are the most frequent causes of this condition; it often, however, originates in congestion, and remains after the hyperæmia has passed away. The rare cases of intermittent aphonia dependent on malarious influences, which have been reported,[1] probably belong to this category. I have once seen it caused by extensive cerebral disorganization from a tumour at the base of the brain.

SYMPTOMS.—The condition is seen with the laryngoscope, on directing the patient to attempt to produce some vocal sound,—that is, to try to say "*ah*" or "*e*;" the vocal cords may not move, or may approach each other only very slightly—in all cases remaining distinctly apart. As the vocal cords remain at the side of the larynx, the condition might be called "bilateral paralysis with lateral fixture." The laryngeal mucous membrane is generally pale. The voice, of course, is always suppressed. It is only the voluntary action of the adductors of the vocal cords which is impaired; the involuntary or reflex movements, especially those of a forcible character, are not generally affected. Thus, coughing and sneezing are usually accompanied with *sound*, showing that the cords approximate. In laughing, however, where the expirations are much less forcible, especially in feeble people, there is often no sound. In other words, these patients do not laugh, but only smile, the term "laughter," strictly considered, being an audible manifestation. The constitutional condition is such as has been already indicated under the head of the Causes of the local phenomena.

PATHOLOGY.—The pathology of the disease probably consists in the "nerve-force" being feebly or imperfectly evolved, or not directed in the proper channel: there is no lesion here. The muscles, which are paralysed, are the crico-arytænoidei laterales on both sides, and the arytænoideus proprius.

PROGNOSIS.—The prognosis is very favourable in almost all cases.

TREATMENT.—The treatment consists in the use of local remedies which tend to excite approximation of the vocal cords. Stimulant solutions were formerly recommended, but Faradization is a far more effective remedy. One pole should be applied over the thyroid cartilage externally, the other one to the vocal cords. My "laryngeal electrode"[2] will be found very useful. With it, I have cured aphonia of eight, and even ten years' standing. The instrument has

[1] Valleix, Bulletin de Thérapie, 1843.
[2] Made by Mayer, 59, Great Portland Street, W.

been also successfully used by other laryngoscopists, both in this country and on the Continent. The patient's health may generally be benefited by constitutional (tonic) remedies.

Unilateral Paralysis of the Adductors of the Vocal Cords.

DEFINITION.—A condition in which, owing to one of the vocal cords not being drawn to the median line on attempted phonation, there is loss of voice.

CAUSES.—The condition may be caused by local injuries, may occur in chronic toxæmia (lead and arsenic), or may be due to cerebral disease, or pressure on the pneumogastric or its recurrent branch. It is difficult to say whether its occurrence as a sequel of diphtheria is due to the first or second cause.

SYMPTOMS.—The condition is seen with the laryngoscope. On attempted phonation one vocal cord remains at the side of the larynx, whilst the other is drawn to the median line. The condition may be described as "unilateral paralysis with lateral fixture." The mucous membrane covering the affected cord is generally congested. There is aphonia or dyspnœa, and usually an absence of constitutional symptoms. When the paralysis is complete, or even much marked, the acts of coughing, sneezing, and laughing are always altered in character, and often unaccompanied by sound: indeed, a modification of the natural cough or sneeze is sometimes one of the earliest symptoms of the condition. When the unilateral paralysis is accompanied with loss of power of the same side of the tongue and palate, it indicates serious cerebral disease near the nucleus of the spinal accessory nerves.[1]

PATHOLOGY.—As regards the pathological anatomy, I may observe, that in the only case of this disease—a case of seven years' standing, which I have examined after death, there was considerable atrophy of the crico-arytænoideus lateralis on the affected side. The arytænoideus proprius did not seem to suffer. The disease is probably often due to inflammatory exudation, either of a simple or dyscrasic character, into the substance of the muscle. When the pneumogastric nerve or its recurrent branch are pressed upon, the abductor muscle is always so much more affected than its antagonist, that the function of the adductor seems to be little affected.

PROGNOSIS.—The condition not being in itself dangerous, and being generally due to local causes, need not, as a rule, give rise to serious apprehensions. If there is evidence, such as paralysis of other parts, to

[1] See Dr. Hughlings Jackson's valuable Illustrations of Diseases of the Nervous System. Lond. Hosp. Reports, vol. i. p. 361, and vol. ii. p. 330.

show that the disease is due to cerebral causes, the prognosis is, however, serious. It is always very difficult to cure.

TREATMENT.—This should be the same as that recommended for bilateral paralysis of the adductors; it is not, however, generally so successful. When resisting the action of my ordinary electrode, the "No. 3 laryngeal electrode," by means of which one pole can be passed into the larynx and the other into the hyoid fossa, so that the current passes through the crico-arytaenoideus lateralis, may be employed. Constitutional remedies may be used with advantage in cases of chronic toxæmia.

Bilateral Paralysis of the Abductors of the Vocal Cords.

DEFINITION.—A condition in which, owing to the vocal cords not being drawn aside (but remaining fixed near the median line) in inspiration, there is great dyspnœa and stridulous breathing, without much alteration in the character of the voice.

CAUSES.—The causes of this condition are generally cerebral, but morbid influences which affect both pneumogastric or both recurrent nerves may give rise to it. In a case of ex-ophthalmic goitre, I once saw it caused by an enlarged and constricting thyroid gland, which passed round the trachea and pressed on both recurrent nerves; scrofulous deposit in the bronchial and cervical glands, especially in children, is apt to give rise to it. In cancer of the œsophagus, when the deposit affects the anterior wall of that tube, both the recurrents may be involved. It is, however, most commonly caused by central disease of the nervous system. It sometimes depends on simple degeneration of the muscles, without there being any evidence of implication of the nerves. The condition is fortunately very rare.

SYMPTOMS.—On making a laryngoscopic examination, the vocal cords do not separate at all on inspiration. There is a slight interval between them, which alters very little, except in forced expiration, as when the vocal cords approximate completely. As the vocal cords always remain near the median line in this form of paralysis, it might be called "bilateral paralysis with central fixture." The vocal cords are generally slightly congested. The voice is usually but little affected, but it may be rather harsh: if the patient does not move at all, the respiration may be little affected; the least exertion, however, brings on dyspnœa and stridulous breathing. The cough is croupy. The condition is in itself apt to produce constitutional symptoms—such as wasting and febrile excitement; and it is often accompanied by paralysis of other parts, or by the cachexia of the disease which indirectly causes it. In children, it produces the symptoms of laryngismus stridulus, and Dr. Ley considered that laryngismus was always of a paralytic nature, and always

due to the same cause,—namely, pressure on the recurrent nerves. True laryngismus depends on other causes which operate in an opposite way.[1] The paralysis of the abductors of the vocal cords, which produces symptoms of laryngismus, is found in children of a more advanced age than those who are attacked by the ordinary form of laryngismus—that is, by spasmodic laryngismus; of course, however, the paralytic form may also occur to the youngest infants. It differs also inasmuch as the symptoms do not completely pass away; exacerbations may occur, but there is always a certain amount of constant stridor and dyspnœa.

PATHOLOGY AND MORBID ANATOMY.—The pathology of the disease has, to a certain extent, been discussed in considering its etiology. The disease consists essentially in a loss of power of the arytænoidei postici, the powerful abductors of the vocal cords, and is dependent on the interception or non-generation of the nerve current which, through the medium of the pneumogastric and its branches, supplies those muscles in the normal state. In the case of a patient under the care of Dr. Hughlings Jackson in the London Hospital, where I had diagnosed bilateral paralysis of the abductors during life, these muscles, when examined by Mr. Rivington after death, were found to be greatly atrophied. There is probably, generally, also atrophy of the nerve structure.

PROGNOSIS.—The prognosis is very serious both on account of the immediate danger of suffocation implied by the condition, and from its being sometimes an indication of some very serious disease elsewhere, either in the brain or along the trunks or branches of both pneumogastric nerves. The condition is in itself highly dangerous; for though the simple action of the adductors (the abductors being paralysed) is not generally sufficient to close the glottis completely, the addition of a little inflammatory swelling or œdema would soon bring about that state.

TREATMENT.—The operation of tracheotomy should be performed without delay, to save the patient from dying of suffocation. The operation would be likely to exercise a favourable effect on the cerebral disease, for the indirect influence of the exceedingly narrowed glottis (through the respiratory system) on the cerebral circulation must be highly injurious. I cannot recommend any medical treatment either of a local or general character.

Unilateral Paralysis of the Abductor of one Vocal Cord.

DEFINITION.—A condition in which, owing to one vocal cord not being drawn aside (but remaining near the median line) on inspiration,

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[1] See Spasm of the Vocal Cords, p. 448.

there is some dyspnœa and stridulous breathing, without much alteration in the character of the voice.

CAUSES.—The causes which lead to paralysis of one abductor are the same as those which produce the bilateral form of paralysis, but the condition now under consideration is more often due to peripheral causes; that is to say, to pressure on one pneumogastric or one recurrent nerve. Aneurisms of the arch of the aorta by pressure on the left recurrent nerve not unfrequently produce this kind of paralysis of the left vocal cord;[1] and in the year 1866 a case of aneurism of the right carotid occurred in my practice, in which the right vocal cord was paralysed. Cancerous tumours occasionally involve the pneumogastric or its branches, and the strumous glands along the trachea may do so likewise. In malignant stricture of the œsophagus, when the disease affects the anterior wall of that tube, one of the recurrent nerves is occasionally affected.

SYMPTOMS.—The condition can be observed with the aid of the laryngoscope; on directing the patient to inspire, the affected vocal cord is not drawn to the side as in the normal state; its inner edge, however, is not quite in the median line. The vocal cords are generally congested. The condition may be described as "unilateral paralysis with central fixture." There is stridulous breathing and dyspnœa on the slightest exertion, but the two last symptoms, as might be expected, are not quite so severe as where both cords are affected. The constitutional symptoms vary with the different conditions which give rise to this form of paralysis, but this kind of glottic obstruction generally after a time causes symptoms of irritative fever.

PATHOLOGICAL ANATOMY.—The immediate nature of the disease and condition of the nerves and muscles is the same as that which is found in bilateral paralysis with central fixture, but here the disease only affects one side. In several cases which I have brought before the Pathological Society of London,[2] the muscle of the affected side has been seen to be completely wasted—only a few of its inner and lower fibres remaining, whilst its fellow on the opposite side was healthy and well nourished. In some of these cases, the left recurrent nerve has been so completely incorporated in a cancerous or an aneurismal tumour, that its course (after entering into the tumour) could not be traced.

PROGNOSIS.—The condition is generally indicative of very serious disease elsewhere, and the most unfavourable opinion should be given as to the prospects of the case.

TREATMENT.—There is generally little to be done towards the cure

[1] Med. Times and Gaz. January 1864, and Pathol. Transactions, vols. xvii. xix. and xxi.
[2] Pathological Transactions, vols. xvii. xix. and xxi.

of the disease : tracheotomy should be performed if the symptoms of suffocation are at all urgent.

VARIETIES.—In addition to the more palpable forms of paralysis which on the one hand produce aphonia, and on the other lead to suffocation, there are certain states in which loss of power is manifested, by the inability to produce certain notes in singing. Here the crico-thyroid or thyro-arytenoid muscles—muscles, the action of which, though generally supposed to be antagonistic, is probably, in point of fact, co-ordinate—are generally at fault. The limits of this article render a detailed handling of this difficult subject impossible ; but for further details the reader is referred to my pamphlet on the subject.[1] The prognosis, as regards cure, must depend on the age of the patient, the duration of the condition, and the natural character of the voice (whether it be tenor or bass). The treatment must sometimes be stimulant (electricity, astringent solutions, &c.), at other times sedative.

Spasm (or Spasmodic Approximation) of the Muscles of the Vocal Cords.

DEFINITION.—A condition in which there is sudden temporary complete or incomplete approximation of the vocal cords, characterised in the former case by arrest of the respiratory movements and apnœa, in the latter by stridulous inspiration and dyspnœa.

SYNONYMS. — *Latin*—Laryngismus stridulus, Laryngitis stridula, Spasmus glottidis, Cynanche stridula, Cynanche trachealis spasmodica, Asthma Koppii, Asthma Millari, Asthma intermittens infantum, Asthma thymicum; *French*—Laryngite striduleuse, Faux croup, Pseudo-croup nerveux, Spasme de la Glotte; *German*—Kehlkopf-krampf, Stimmritzenkrampf (Cerebral Croup, Pseudo-croup); *English*—Millar's Asthma, Crowing Inspiration, Child-crowing, Spasm of the Glottis, Spasmodic Croup, Spurious Croup, Cerebral Croup, &c. &c.

Causes.—The causes of spasm of the vocal cords are involved in a considerable amount of obscurity, and there is evidence to show that many influences may be concerned in its production; hence it is not surprising that the etiological features concerning it should have undergone various changes and modifications.

The causes may be divided into (1) central, and (2) peripheral—the latter being subdivided into (*a*) direct, and (*b*) reflex.

1. The disease was at one time considered to be always dependent on cerebral disease, or at least on a disordered state of the functions of the brain,[2] and this view, which has been assailed in various ways, seems to be again gaining ground. Numerous cases are on record, where

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[1] Hoarseness and Loss of Voice. Churchill, 1868.
[2] Commentaries on Diseases of Children, by Dr. John Clarke.

other admitted symptoms of cerebral disease manifested themselves before the occurrence of laryngeal spasm. Limited congestion or interstitial exudation of serous fluid, near the origin of the pneumogastric nerves, is probably the condition of the brain which is concerned in the production of this phenomenon. In many cases, however, the structural alteration of the brain, if present, is of too delicate a nature for detection, and still more frequently a morbid condition of that organ is produced by the sudden apnœa. Hence, even when the brain is the primary seat of the disease, it is impossible to speak with certainty as to the nature of the morbid condition. The cerebral affection is probably often dependent on a dyscrasic state.

A rachitic condition of the bones of the skull has frequently been noticed. Out of ninety-six cases of laryngismus examined by Lederer, there was craniotabes in ninety-two.[1] The experience of Sir William Jenner and Dr. Wiltshire is of a similar character.[2] It has been suggested that the thinness of the cranial bones in rickets allows pressure to be exercised on the brain in the occipital region, when the child lies on its back (Elsässer); but it is more probable that the rachitic dyscrasia is accompanied by morbid changes of a nutritive character in the structure of the brain itself.

Scrofula has also been regarded as an active predisposing cause of the disease (Marsh). Sometimes an attack is brought on by tossing the child in the air, and it still more often comes on in sleep. These facts have been adduced by some as an evidence of the cerebral nature of the disease; but it must be remembered that both in sleep and in sudden movements of the body the function of respiration not less than the cerebral circulation is modified, and that the spasm of the glottis may originate in either process. Disease of the cervical portion of the spinal cord sometimes gives rise to it (Marshall Hall). In cases of disease of the brain or medulla, external pressure applied over these parts has been known to cause laryngismus. Hydrocephalus exists in some cases; and mental emotion—especially terror and rage—occasionally gives rise to the spasm.

2. (a) Direct pressure on the recurrent or pneumogastric nerves by enlarged and tuberculous cervical and bronchial glands has since Dr. Ley's time been regarded as a cause of laryngismus, but in these cases the cause is probably (as Dr. Ley conceived) "paralysis of the dilators of the glottis." Enlargement of the thymus gland was at one time, especially in Germany, considered the essential cause of laryngismus,[3] but at present its influence is considered to be of a very exceptional character. In so far as these causes operate by producing paralysis of the abductors of the vocal cords they belong to the last section of neuroses, but they probably often cause spasm of the adductors by obstructing the venous circulation through the neck, and thus giving rise to cerebral irritation.

[1] Rühle, Kehlkopfkrankheiten, p. 201. Berlin, 1861.
[2] See art. Rickets, vol. i. p. 786.
[3] Kopp, Denkwürd. in der ärzt. Prax. Frank. 1820.

(b) Amongst the reflex causes of spasm we have those
directly on the larynx and those operating at a distance. A
not unfrequently come on whilst the child is sucking or rather
lowing, and there can be little doubt that the cause here is the p
of liquid into the larynx. Spasm produced by dangling the ch
the air is probably caused by the impression of a current of air
glottis. Amongst the reflex causes of laryngismus which ac
distance, there is the irritation of teething, the presence of indig
food or helminthoid parasites in the alimentary canal, and the in
sion of currents of cold air on the integument. It sometimes
venes on the cure of a protracted diarrhœa or a chronic skin aff
but these causes probably act by setting up cerebral irritation.
been noticed by Sir William Jenner[1] that the mother's health
important influence in the production of rickets, and Kopp has
precisely the same observation with regard to laryngismus. Here
is another link towards the chain of association which Sir W
Jenner has attempted to establish between these two morbid cond
The greater liability of the male sex, which occurs in other lary
diseases, holds good here. The disease is most frequent betwe
ages of six months and two years.

SYMPTOMS.—The age of the patient destroys the value of subj
symptoms, but those of an objective character are sufficiently m
The following is the common history of a first attack. A child
bed, apparently in its ordinary state of health, wakes up sudde
about midnight with difficulty of breathing, inspiration being a
panied by a crowing noise similar to that heard in croup. Aft
or three of these stridulous inspirations, the frightened child
out crying and in a few minutes is fast asleep again, as if nothin
occurred. This description does not apply to every case. The
may have been peevish and fretful for a few days before, may
suffered from loss of appetite, and may have been restless at nig
a slight "catch" in the breath may have been previously n
The first attack may come on at any other time, but it most freq
occurs during sleep. The next day the child may be quite wel
there may be no further return of the symptoms, but it often ha
that another attack comes on about the same hour the following
The second attack is generally more severe than the first, both
character and duration. In severe cases, indeed, the paroxysms
a most urgent kind and of the most frequent occurrence. A
fit of laryngismus may be thus described: the breathing sud
becomes greatly embarrassed, each act of inspiration being muc
longed and accompanied by a harsh stridor: suddenly the
ceases, the glottis is completely closed, and the respiratory move
of the chest are suspended. The flush which first lit up the
tenance gives way to pallor and afterwards to lividity. The ey
roll, the veins of the neck are turgid, the fingers close on the th

[1] Op. cit.

which is bent in the palm, and the hands are flexed on the wrist; spasm likewise affects the feet, the great toe is drawn away from the other toes, and the foot is flexed and rotated slightly outwards. These so-called "carpo-pedal" contractions are probably sometimes accompanied with great pain. The disease, indeed, may partake of the character and assume the form of epilepsy. Notwithstanding the severity of the paroxysm just described, the patient may survive it, the apnœa being succeeded by stridulous breathing, and by relaxation of the spasmodic contractions of the feet and hands; but when the symptoms are of the dangerous character just described, the paroxysm is sure to be quickly followed by others—in one of which the child dies. The severity of the attacks varies between the mild paroxysm which has been described as occurring at the commencement of the disease, and one sufficiently intense to cause death. The spasm is characterised by its sudden occurrence and by its complete remission, as a rule by the entire absence of febrile irritation and by the progressive severity of the spasm, as regards recurrence, duration, and intensity. Some of the associated symptoms of laryngismus may likewise be present, such as hydrocephalus, a rachitic condition, or enlargement of the thymus body.

DIAGNOSIS.—The non-febrile and distinctly intermittent nature of the affection differentiates it from true croup, and its own distinctive characters from all other diseases.

PATHOLOGY.—The pathology of the disease has been considerably encroached upon in considering its causes, but there still remains something to be said concerning its nature. There are two points on which it appears to me necessary to insist: these are (1) that in all cases there is an altered state of the nerve-centres; and (2) that the immediate cause of the phenomena of the stridulous inspiration and apnœa is spasm of the adductors of the vocal cords. The facts which point to an alteration in the brain substance (whether recognisable or not) are first, that both sides of the body (both vocal cords) are affected; secondly, that the various alleged causes (such as dentition, indigestion) are not only often in operation without the production of laryngeal spasm, but when they do give rise to that symptom they necessarily act through the brain; thirdly, that frequently other admitted symptoms of cerebral irritation, such as the carpo-pedal contractions, are present. That the disease depends on spasm of the adductors of the vocal cords appears probable for the following reasons:—(1) The other phenomena are those of spasm (carpo-pedal contractions). (2) Complete closure of the glottis never takes place under physiological conditions, and therefore it is improbable that the simple action of the adductors of the vocal cords could cause complete closure of the glottis (the action of the abductors being in abeyance); in support of this view I may observe that in three cases of paralysis of the crico-arytænoideus posticus which have come under my notice, the inner edge of the affected cord was not adducted to the median line. (3) The total

remission of so urgent a symptom points to its cause being of a spasmodic nature ; there being, as far as I am aware, no instance of complete paralysis of a truly paroxysmal character.

PROGNOSIS.—The prognosis depends on the character of the paroxysm and its supposed cause. The cases mainly dependent on reflex causes (dentition or irritation of the alimentary canal) generally do well, whilst those due to direct pressure and those mainly caused by cerebral irritation are more frequently fatal. Thymic asthma is especially dangerous, and if there is evidence (such as considerable enlargement of the gland) to show that the spasm is of that character, the most unfavourable opinion must be given. The length of the intervals between the paroxysms is a good prognostic guide—the longer the interval, the better the chance of recovery.

TREATMENT.—The treatment must be twofold : first, to relieve quickly the spasm of the glottis ; secondly, to attack the source of the disease. The immediate treatment generally falls to the nurse or mother. The little patient should be raised and placed in a sitting posture, and then he may be slapped on the back, cold water may be dashed in the face, and ammonia or strong acetic acid held to the nose. These measures are often successful by giving rise to violent expiratory actions ; but remedies calculated to relieve spasm are equally successful. The warm bath may be used and emetics given directly there is a sign of the stridor—when the paroxysm is on, the child will not drink. A favourite remedy in Germany, and one that is highly successful, is tickling the fauces with the finger or a feather until vomiting is produced. Depressing enemata, such as tobacco, have likewise been recommended, but their use is attended with considerable danger. The ordinary rules for the treatment of disease apply here ; that is to say, gentle remedies should be used in mild cases and those of a more powerful character in dangerous ones. Putting the lower part of the child's body in a hot bath and dashing cold water in its face is a simple and sometimes successful plan. The inhalation of chloroform is a very valuable remedy, but of course must be used with great care, and cannot safely be employed by non-professional persons. If the child appears to be sinking from the apnœa, the trachea must, of course, be opened, and artificial respiration resorted to. Indeed this should even be adopted by the practitioner, should he arrive shortly after the apparent extinction of life. Some practitioners recommend the use of antispasmodic remedies (whether animal, vegetable, or mineral) between the fits. As regards the *fons et origo mali*, the most suitable treatment will be found detailed in the various articles in these volumes which treat of scrofula, rickets, hydrocephalus, dentition, parasites, &c. Enlargement of the thymus must be treated by the application of leeches (according to the age and strength of the patient), and afterwards by counter-irritation.

VARIETIES.—The ordinary kinds of laryngismus, according to my views, are essentially due to spasm of the adductors of the vocal cords, but that variety which is caused by pressure on the pneumogastric or recurrent nerves is due to paralysis of the abductors. It has been treated of in the last section of neuroses; and differs from ordinary laryngismus, in the ways there indicated. It appears to me that Dr. Ley was right as to the cause of the symptoms of a certain form of laryngismus, but mistaken in regarding a rare variety as a typical example. This view explains the very opposite opinions which have been held concerning the· etiology and pathology of the disease.

I have thought it more convenient to treat spasm of the glottis as an infantile affection, but it must be borne in mind that it sometimes occurs to adults. Women are generally the subjects of it; and it is commonly regarded as an hysterical phenomenon. In one case, however, that came under my notice, there were no symptoms whatever of hysteria, the stridulous inspiration being so much worse during sleep, that the patient, a woman in the London Hospital, was obliged to be placed in a separate room, on account of keeping the other patients awake. In this case, though the rest of the mucous membrane was much congested, the vocal cords were perfectly healthy. The case recovered under the local treatment recommended under the head of Chronic Laryngitis.[1]

The treatment should be the same as that advised for children, though inhalations of sedative and anæsthetic vapours may here be employed with advantage. Spasm of the glottis, dependent on the inhalation of poisonous gases and the impaction of foreign bodies in the œsophagus, requires the most prompt treatment; if not immediately relieved, laryngotomy or tracheotomy should be performed without delay. One form of spasm of the vocal cords is that met with in hooping-cough—the essential phenomena of this complaint being a series of short, rapid, and violent expirations, followed by a prolonged stridulous inspiration—the disease which will be found treated in detail elsewhere.[2] The laryngeal cough, sometimes met with in hysterical women whose larynx is seen with the laryngoscope to be perfectly healthy, is also due to a spasmodic tendency of the adductors of the cords, the spasm only occurring in expiration; and the same may be said of the sharp ringing cough which occasionally affects children, and is usually looked upon as of a reflex nature. The nervous laryngeal cough of adults is as difficult to treat as most hysterical complaints. I have found the most satisfactory results follow from the use of warm sedative and anæsthetic inhalations; but the results are often disappointing. Lasègue reported a case successfully treated by belladonna;[3] but in a severe case that came under my care, atropine was given till its full physiological effects were

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[1] Med. Times and Gazette, Nov. 15, 1862.
[2] Vol. i. p. 271.
[3] Archives Générales, May 1854.

produced, but without relief of the cough. Dr. Harley has reported a case[1] in which valerianate of zinc effected a cure.

DISEASES OF THE SENSORY SYSTEM OF THE LARYNX.

Hyperæsthesia.

Increased sensibility occurring independently of inflammatory disease or structural alteration of the tissues of the larynx, is undoubtedly a rare morbid condition, but it may occur either in an intermittent form or without any periodic character. A case of the former kind is reported by Dr. Gerhardt,[2] and a few of the latter have fallen under my notice. Several cases have also been reported by Dr. Handfield Jones.[3] Neuralgic cases should be treated on the ordinary principles which regulate the therapeutic management of such cases. The inhalation of hot sedative vapours and anæsthetics does good in cases of a non-intermittent character; and the internal use of narcotics is also indicated.

Some of the morbid phenomena already referred to under the head of Motor Affections (such as pertussis and nervous laryngeal cough), may be due to increased sensibility of the mucous membrane of the vocal cords—the hyperæsthesia manifesting itself in reflex action.

Anæsthesia.

Though there is great difference between the sensibility of the glottis in different people, anæsthesia rarely occurs as a distinct morbid affection.

Disease affecting the origin or trunks of the pneumogastric nerves or their superior laryngeal branches, would be likely to diminish the sensibility of the larynx in proportion as the function of the nerves was interfered with.[4] Romberg has observed that in cholera there is impaired sensibility of the mucous membrane of the larynx.[5] Some morbid phenomena of a functional character, such as a vocalist's inability to produce certain notes which previously could be easily formed, are probably in some cases (where the larynx appears healthy) due to impaired muscular sensibility.

[1] Med. Times and Gazette, vol. ii. p. 116.
[2] Virchow, Archiv xxvii. Heft 1 and 2.
[3] Med. Times and Gazette, May 2, 1863.
[4] Hufeland's Journ. der pract. Heilkunde, Feb. 1853.
[5] Ibid. Feb. 1832.

SECTION II.

Secondary Diseases of the Larynx in Acute Affections.

SMALL-POX.

The laryngeal affection may be a mild papular or pustular eruption of the mucous membrane, or it may be a severe inflammatory disease accompanied by the presence of false membrane. The former, as a rule, causes little or no inconvenience; the latter is often fatal. In the year 1863, through the courtesy of Mr. Marson, the author of the able article on Small-pox (vol. i. p. 432), I was enabled to examine several patients in the Small-pox Hospital, with the laryngoscope. In one patient labouring under severe purpuric small-pox, I found ecchymose spots on the under-surface of the epiglottis, and on the mucous membrane over the arytenoid cartilages. In a convalescent case, there was a distinct pustule on the edge of the epiglottis; in another case, in which the entire body was covered with pustules, the larynx appeared perfectly healthy; and in another similar case there were no pustules, but there was marked congestion of the mucous membrane; in another case, the upper surface of the epiglottis was covered with pustules. Rühle, who in a bad epidemic of small-pox, in Greifswald, in 1856-57, made no less than fifty-four post-mortems, observes,[1] "Although I have seen here and there pustule-like elevations, I nevertheless consider the essential peculiarity of the laryngeal affection to be of a croupous or diphtheritic inflammation." Dr. Rühle further observes, that, as "out of the fifty-four cases there was not a single case in which the larynx and windpipe were in a normal state, he cannot but attribute a certain proportion of the mortality to the laryngeal affection." Pathological examples of the diphtheritic complications of small-pox are to be found in the museums of St. Thomas's and St. Bartholomew's Hospitals, and in other collections. In two instances, I have known permanent paralysis of the adductor of a vocal cord follow small-pox: in both, the larynx was affected at the time, and it is probable that the affection was of the diphtheritic character. As regards treatment, it may be observed that in the pustular form of the disease interference is unnecessary, and that in the diphtheritic form it is almost useless: in the latter case, however, the local treatment elsewhere recommended for primary diphtheria can be adopted.

MEASLES.

In this disease the affection of the larynx may be either a simple catarrh, or a severe croupous affection.

[1] Op. cit. p. 247.

The catarrhal form of laryngitis may occur before the eruption appears, a day or two after the rash has come out, or when it is beginning to decline. It is more common than the croupy form of disease; and though occasionally the inflammation runs high, it is seldom of any importance. In some epidemics, catarrhal laryngitis comes on when the eruption has almost disappeared.[1] In these cases, there is generally obstinate hoarseness. In a number of Professor Hebra's patients in the General Hospital at Vienna, in different stages of measles, Dr. Stofella [2] found a highly-injected condition of the mucous membrane of the larynx in almost all the cases which he examined laryngoscopically.

The croupy or diphtheritic form of inflammation, observes Dr. West, "seldom begins until the eruption of measles is on the decline, or the process of desquamation has commenced. Its appearance is most frequent from the third to the sixth day from the appearance of the eruption, but it oftener occurs at a later than an earlier period."[3] The treatment should be similar to that recommended (vol. i. p. 261) for primary croup, but it must always be borne in mind that the secondary disease is of a less sthenic type.

SCARLATINA.

The affection of the larynx in these cases may be either an acute œdema of the glottis or a croupous inflammation: they are, fortunately, both rare complications. The œdema which sometimes occurs in scarlet fever may be dependent on the debility which exists during the convalescence of severe febrile complaints, or may be due to the renal affection which sometimes follows scarlatina.

The croupy inflammation of the larynx, though not common, is peculiar to some epidemics. Goupp described an epidemic in Wurtemberg, in which, in the greater number of cases, croupy symptoms appeared from the third to the fourth day of the illness; in some cases death took place before the exanthem appeared.[4] It has been observed, that in diseases of the larynx dependent on, or associated with, scarlatina, there is a great tendency to the ulcerative process. A specimen (No. 36, series W), in the Museum of St. Thomas's Hospital, supports this view. The larynx was taken from an adult patient, who died of scarlatina: there is a very thin layer of lymph covering the mucous membrane of the larynx, and the right arytenoid is laid bare by a large ulcer.

The treatment of the plastic form of inflammation should be such as is recommended for diphtheria, viz. the internal use and local application of the persalts of iron, a highly nourishing diet, and the free use of alcoholic stimulants, well diluted. The practitioner must always

[1] Bohn, Konigsberg Mediz. Jahrbücher, 1858.
[2] Wien Medizin. Wochenschrift, Nos. 18, 19, 20, 1862.
[3] Diseases of Infancy, p. 236.
[4] Rühle, op. cit. p. 243.

have tracheotomy in view. In œdema, this operation is also likely to be necessary, but scarification should be first tried.

ERYSIPELAS.

In erysipelas of the head and neck there is always more or less congestion of the mucous membrane of the larynx; and even when the erysipelatous inflammation is seated on the limbs, there is sometimes sympathetic or concomitant inflammation of the larynx. It sometimes, though less frequently, occurs in hospital gangrene.[1] It may result in an acute œdema, which rapidly tends towards a fatal termination. The symptoms of the disease are, difficulty of swallowing, hoarseness or loss of voice, and pain; the latter is increased on pressure. Dr. Semeleder has examined five cases with the laryngoscope; in four of them the erysipelas affected the face, and in these he found inflammatory redness and swelling of the epiglottis and larynx down to the vocal cords, though there was no dyspnœa or dysphonia. The inflammatory symptoms in the larynx disappeared gradually with the desquamation of the skin; and in one case a relapse of the cuticular affection was accompanied by a recurrence of laryngeal inflammation. In the fifth case—erysipelas of the lower extremities—there was no hyperæmia of the larynx. The poison of erysipelas sometimes confines itself to the larynx, the skin being free from inflammation; at other times it passes from the larynx to the external parts. Cases are on record, at least, which tend to support these views.[2]

The treatment should be active, and such as has been recommended in ordinary inflammation and œdema of the larynx.

TYPHUS AND TYPHOID.

In *typhus* there is nothing characteristic about the laryngeal affection; congestion of the mucous membrane, plastic deposit on its surface, gangrenous inflammation, and œdema, being conditions which are all occasionally met with. The ulceration is generally of the most destructive character, and whilst it often involves a large surface, it frequently penetrates deeply and exposes the cartilages. It is generally at the posterior parts of the larynx, that is, *at the under part*, in the prone position of a patient with low fever, that the disease is most frequently found; and it is commonly thought to be caused, at least in part, by hypostatic influences. The cricoid cartilage is frequently seen to be denuded, and of a blackish grey colour, and there is frequently a corresponding discoloration of the opposite wall of the pharynx.

In *typhoid* the same conditions are met with as in typhus; but there seems to be a greater liability to œdema, the ulcerative process

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[1] Ryland, Diseases of the Larynx, p. 8.
[2] Ibid. pp. 73 to 77.

more often appears to originate in a typhous deposit,—"laryngo-typhous being, as it were," says Rokitansky, "the completion of abdominal typhous;" and it is said that the cartilages often become independently diseased, *i.e.* become diseased without the superjacent tissues being primarily affected. So many conditions of the larynx are met with which tend to lead to destruction of the cartilages, that it seems unnecessary to resort to the theory that these structures become independently diseased. If in cases where the cartilages are affected the patient survives the fever, the pathological changes described at page 461 take place, and the case runs the course of laryngeal phthisis. Dr. Wilks has especially called the attention of the profession in this country to the ulceration of the larynx occurring in typhoid.[1]

TREATMENT.—In these cases, where subjective symptoms are often altogether absent, and those of an objective character are to a great extent masked, the dictates of rational medicine should lead us to be prepared by surgical interference (tracheotomy) to prevent death from laryngeal obstruction, rather than to attempt to control or oppose the disease.

SECONDARY DISEASES OF THE LARYNX IN CHRONIC AFFECTIONS.

LARYNGEAL PHTHISIS.

DEFINITION.—Chronic thickening and ulceration of the larynx, usually occurring consecutively to pulmonary phthisis, but sometimes being present before there is any evidence of lung-disease. There is hoarseness or loss of voice, often dysphagia and dyspnœa, with persistent increase of temperature, and continuous wasting of the body.

SYNONYMS.—*Latin*—Phthisis laryngea, Laryngitis chronica, Tuberculosis laryngis, Laryngophthisis, Helcosis laryngis; *French*—Phthisie laryngée; *German* — Kehlkopftuberculose; English — Laryngeal Phthisis, Throat Consumption.

CAUSES.—The causes are the same as those which give rise to other laryngeal affections (such as exposure to cold, functional excesses, &c.), *plus* a special constitutional condition, either inherited or acquired, through which cell-proliferation takes place in the sub-mucous tissues. In ordinary chronic laryngitis the rapid evolution of imperfect cells takes place at the free surface, but in laryngeal phthisis the interstices of the tissue are the seat of the deposit. Although Niemeyer has done good service in so decisively combating the idea of the *tubercular* origin of all forms of phthisis, and in pointing out the catarrhal and inflammatory nature of many cases of that disease, there can be no doubt that a disposition to low interstitial inflammation is often

[1] Transact. Pathol. Soc. vol. ix. p. 34, and vol. xi. p. 14.

inherited, or, at any rate, congenital. The feeble texture is excited to chronic inflammation and cell-proliferation by very slight exciting causes. By the Vienna school, the cell-proliferation was called an "exudation," and probably, in a large number of cases, the deposit is more of this nature than that of a true *growth ;* the weak constitution which gives rise to it was called "a diathesis." Our views on phthisis are now undergoing a great change, but however unimportant a *rôle* tubercle may play, that there exists a diathetic predisposition to low inflammatory action cannot be denied. Laryngeal phthisis is often hereditary, and it frequently attacks several brothers and sisters ; as in other laryngeal affections, males show a greater proclivity to it than females.

Numerous cases of laryngeal phthisis have come under my inspection, where the most experienced stethoscopists have been unable to discover a trace of lung-disease ; but, on the other hand, I must admit that I have only three times met with cases of laryngeal phthisis in the dead subject without finding corresponding pulmonary disease.

SYMPTOMS.—*Subjective.*—There is nothing characteristic about the subjective symptoms : they resemble those met with in chronic laryngitis, except that, owing to the thickening of the tissues, the act of deglutition is more often performed with difficulty. Pain is sometimes experienced in swallowing, but it more often happens that the act is difficult—violent coughing coming on from a little food getting into the larynx ; sometimes the drink is violently ejected through the nares. The difficulty of swallowing is most extreme when the epiglottis is much thickened ; but it also generally occurs when the ary-epiglottic or inter-arytenoid folds are much swollen.

Objective.—(1) *Vocal.*—Dysphonia, or aphonia, are always present ; hoarseness being generally the symptom of the early stages, complete aphonia of the later. The aphonia is, of course, generally dependent on structural changes, but it may occur at the commencement of the disease from functional causes (weakened approximative action of the vocal cords, and feeble action of the expiratory muscles). The cough varies in different stages. Sometimes the disease is ushered in with violent and frequent paroxysms of cough which nothing can alleviate ; sometimes it is only an occasional dry tickling cough ; it is generally aphonic in the later stages of the disease.

(2) *Respiratory.*—The respiration is at first little affected, but afterwards it becomes embarrassed, and inspiration is often stridulous ; mucous râles can generally be heard over the thyroid cartilage and trachea. In the last stage the dyspnœa is so great that tracheotomy occasionally becomes necessary.

(3) *Laryngoscopic Signs.*—In cases of pulmonary phthisis pallor of the mucous membrane is often noticed, and Dr. Semeleder regards anæmia of the larynx, where there is no other cause for its existence, as of some prognostic value with regard to phthisis. Congestion of the mucous membrane is generally the cause of the hoarseness in the

early stages of laryngeal phthisis. At this period there is nothing to distinguish the condition from ordinary chronic laryngitis; when, however, exudation takes place, the appearance is characteristic.

The ary-epiglottic folds look like two large, solid, pale, pyriform tumours, the large ends being against each other in the middle line, and the small ones directed upwards and outwards. The surface is, as remarked, generally pale, but there may be accidental congestion. The inter-arytenoid fold is absorbed in these swellings, which interfere with the action of the arytenoid cartilages, and thus prevent the approximation of the vocal cords. Sometimes the swelling only affects the ary-epiglottic fold of one side, and at first the projection of the cartilages of Wrisberg and Santorini interfere with the distinctly pyriform shape of the tumours, but when developed they are pathognomonic of the disease. The condition described is really only chronic œdema of the ary-epiglottic folds, but when once fully established it is as certain to terminate fatally as a case of acute tuberculosis or encephaloid cancer. Its course is, of course, not so rapid as that of the diseases mentioned, but the end is similar. The epiglottis is not unfrequently thickened; sometimes it is so much enlarged as to prevent an inspection of the parts below. Its shape is often somewhat turban-like, the normal contour and surface marks having completely disappeared. In addition to the thickening, the epiglottis is in fact often rolled backwards on itself, so that the free edges cannot be seen in the laryngeal mirror; in other cases, where they are visible, the cartilage is often exposed from ulceration. Thickening and ulceration of the posterior part of the ventricular bands (false vocal cords) can sometimes be seen, but disease may make considerable progress in this part without coming into the field of vision. Ulceration of the vocal cords is not unfrequent, the most common situation being at the *processus vocalis*, the junction of the cartilaginous and ligamentous portions.

(4) *Miscellaneous Symptoms.*—The laryngeal secretion varies greatly both in quantity and quality, and probably depends more upon the condition of the bronchial tubes and lungs than upon that of the larynx. The constitutional symptoms are those of pulmonary phthisis. The course of the symptoms varies with the site of the disease, the progress being most rapid when the epiglottis is affected, and generally much slower when the ary-epiglottic folds are the parts implicated. The termination is nearly always fatal, where thickening has taken place to any considerable extent.

DIAGNOSIS.—Where the characteristic pyriform swellings of the ary-epiglottic folds are present, it is impossible to mistake the disease; but where the thickening is not of such a defined character, the diagnosis is not quite so clear. The only conditions which are likely to give rise to an error are acute œdema, and syphilitic thickening. In acute œdema, the rapid occurrence of the disease and the transparent character of the swelling differentiate it, and in syphilis the thickening is not considerable, whilst the ulcerative process is more active.

PATHOLOGY.—It is difficult to investigate the pathology of laryn-geal phthisis, because of the close mutual interdependence of the conditions of the larynx and lungs. As the result, however, of careful observation with the laryngoscope in a great number of cases, the ordinary course of events appears to me to be as follows :—1st. There is chronic hyperæmia, generally of a higher degree but more limited ex-tent than is met with in ordinary chronic laryngitis. 2dly. Thickening of the tissues takes place, the kind of thickening varying in different parts ; thus, the epiglottis and vocal cords appear to become infiltrated with a semi-solid material, whilst the ary-epiglottic folds become dis-tended by a simple serous exudation ;[1] the thickening of the ventri-cular bands (false vocal cords) is generally of the solid character, but is occasionally serous. 3dly. Small ulcers form ; these afterwards coalesce and produce larger ulcers (the secondary tubercular ulcers of the larynx of Rokitansky). The small primary ulcers, which are fre-quently first seen at the posterior extremity of the ventricular bands and on the under-surface of the epiglottis, when watched with the laryngoscope, often appear to commence in the minute racemose glands. Subsequently the ulceration spreads to other parts : some-times, however, the ulcerative process commences in the vocal cords—destruction of epithelium often occurring some time before the dense structure of the cord itself is affected. In other words, when the cords are first attacked, denudation of epithelium precedes deposit in the tissues. The actual loss of substance which takes place in laryngeal phthisis is not generally great, but chronic disease of the cartilages is frequently found when the disease has existed for a few months; and it appears to me that Dr. Addison's dictum that "inflam-mation constitutes the great instrument of destruction in every form of phthisis" is true in this instance. Tubercle appears to play a very secondary part, if any part at all. As regards the relation of laryngeal phthisis to pulmonary phthisis, as already observed, I do not consider that the laryngeal affection is caused by the disease of the lungs. As a rule, the pulmonary disease precedes the affection of the larynx; but still, numerous cases occur in which congestion and thickening of the larynx with hoarseness and cough are found before any disease of the lungs can be detected either by auscultation or microscopic examination of the sputa. On the other hand, in the progress of the disease, evidence of pulmonary disease becomes mani-fest, and I have only met with three cases in which on post-mortem examination laryngeal phthisis was present without any disease of the lungs. As an almost invariable rule, cavities are found in the lungs, or at least breaking down of lung tissue.

MORBID ANATOMY.—On examining the larynx of a patient who has died from laryngeal phthisis, there is commonly found great thickening

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[1] In 14 of the 274 cases of œdema of the glottis collected by Sestier (Traité de l'Angine œdématense ; Paris, 1852), the patients suffered from "chronic laryngitis with pulmonary tubercles."

of the sub-mucous tissues of the larynx, with ulcers varying
from a pin's point to a shilling. The small ulcers are mos
monly found on the under-surface of the epiglottis; the larger
the root of the epiglottis, the posterior extremity of the vent
bands, and at the *processus vocalis*. Sometimes the ulcerative
is limited to the minute glandulæ, and under these circumstanc
mucous membrane presents a worm-eaten appearance. Tube
said by Rokitansky[1] to be deposited in the form of grey granu
in the sub-mucous areolar tissue, or to be infiltrated as yellow
matter beneath the mucous membrane—the true tubercular
being rarely found except over the arytænoideus muscle and th
jacent arytenoid cartilages. Rokitansky does not consider the
ening of the epiglottis to be of the true tubercular character.
never seen the grey granulations referred to, and the deposit
tissues has appeared to me to consist of a serous fluid with
compound granule cells, and with molecular and granular matter.
débris may or may not be tubercular, but even at this period
be of more than historical interest to remark that though Louis[2]
ulceration of the larynx in one-fourth of his cases of puln
phthisis, he did not consider that tubercle was ever deposited
tissues of the larynx. The proportion of cellular elements va
different cases and in different parts; in the ary-epiglottic
they are generally very scarce or altogether absent. Pus is som
found diffused through the tissues, but rarely circumscribed, un
be under the perichondrium of the cricoid cartilage.

Caries of the Cartilages—or, as it is commonly called, *necrosis*
cartilages—far more often results from laryngeal phthisis than fr
other diseases together, and it may be regarded as one of the co
sequelæ.

The death of the cartilages is generally believed, and probabl
truth, to originate in inflammation of the perichondrium. [5]After
that membrane is not unfrequently found to be separated fro
cartilage by a quantity of pus, and ossification of the ca
generally precedes its death. The cartilage, with the exception
more or less ossified condition, may present almost a healthy a
ance, or it may be of a dark grey or even black colour. The pr
or absence of discoloration seems to depend on whether there
communication (through ulceration of the tissues) between the
lage and the atmosphere. In those cases where there is ulcer
their surface, and sometimes even their entire thickness, is discol

Sometimes the cartilages are found to be increased in volum
still more rarely they are completely atrophied. The latter con
is figured by Rühle.[3] The necrosed condition of the cartila
generally associated with the presence of serum or pus in the ad
parts of the larynx; the muscles are soaked in the morbid flui

[1] Pathol. Anatomy, Sydenham Soc. Translation, p. 33.
[2] Louis on Phthisis.　　　　　　　　[3] Op. cit. Plate I.

the areolar tissue irregularly distended by it. The etiological relations between the sero-purulent effusions and the necrosis of the cartilages are of a doubtful, and probably of a varying, character; in some cases the former seem to depend on the latter, while in others the opposite relation appears to exist. Sometimes the effusion occurs in the parts external to the larynx, especially when the cartilages near the surface externally (such as the anterior parts of the thyroid and cricoid) are affected, and there a laryngeal fistula may be produced. According to my experience, the arytenoid cartilages are the most frequently affected next to them the cricoid, and then the thyroid cartilage; it is, however, commonly stated that they are affected in the following order of frequency: first the cricoid, secondly the thyroid, and thirdly the arytenoids.

PROGNOSIS.—The prognosis is of the most unfavourable character. Where the epiglottis is much thickened, the progress of the case is generally rapid; on the other hand, when the disease is limited to the ary-epiglottic folds, its course is usually chronic. The result of carefully watching with the laryngoscope, during the last ten years, a great number of cases of laryngeal phthisis, has convinced me that when once thickening to any extent has taken place, that is, when once the disease is fully established, nothing curative can be effected by treatment. Out of several thousand cases, I have only seen two patients recover. Of course, however, suffering may be mitigated, and life prolonged.

THERAPEUTICS.—The plan recommended for chronic laryngitis sometimes gives relief—the application of mineral astringents, by diminishing the irritability of the mucous membrane, often relieving the troublesome cough. Hot and anæsthetic inhalations likewise sometimes comfort the patient; and in cases accompanied by excessive expectoration, I have seen the secretion completely controlled by the inhalation of an atomized solution of tannin (gr. v. ad fl. oz. j).

It is important to bear in mind that there is a tendency to death in three ways—first, by suffocation, the calibre of the laryngeal canal becoming greatly diminished; secondly, by inanition, the dysphagia being caused by the thickening of the epiglottis and other parts concerned in the act of deglutition; thirdly, by the marasmus, which is a characteristic feature of the constitutional malady; and fourthly, by the combined effect of these influences. The fatal termination may, therefore, be postponed by the performance of tracheotomy, when that operation becomes necessary; by feeding the patient with an œsophageal[1]

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[1] This instrument, which has been provided for me by Messrs. Khrone and Sesseman, consists of a gum-elastic catheter, about 12 inches long, which is connected with an ordinary pear-shaped india-rubber bottle (provided with a tap) by a bayonette joint. The tube is first passed just beyond the larynx, then the bottle (previously filled with a nutritive fluid) is attached, and the fluid injected. The feeding can be effected with a common catheter and an ordinary india-rubber injecting bottle, but this plan does not answer so well.

tube, when normal deglutition cannot be effected; and by the employment of suitable remedies (medicinal and hygienic) against the constitutional debility. It is unnecessary to make any remarks concerning the operation of tracheotomy, as the conditions which render its performance necessary are sufficiently evident. With regard to the use of the œsophageal tube, however, a few observations are called for. The dysphagia, it must be borne in mind, is due more to the act of deglutition being imperfectly performed from non-closure of the larynx by the epiglottis, than by the obstruction in the food tract, caused by the thickened epiglottis. It is from food "going the wrong way," not from the fact of its being prevented passing down the gullet, that the difficulty in swallowing arises. Hence there is generally very little difficulty in introducing the œsophageal tube, especially if it be provided with a duck-billed extremity, and be employed with the aid of the laryngoscope. The fatal termination of phthisis is, of course, much accelerated if the supply of food is to a great extent cut off; and I may observe, that I have prolonged life for many weeks by giving a patient food and stimulants in this way. Alcoholic liquids, which the irritability of the throat would not allow to pass, can be readily introduced into the system by this method. Nutritive enemata can be employed instead of the œsophageal tube, but the results have appeared to me much less satisfactory. Where the patient can swallow a little, but experiences difficulty in doing so from the food occasionally entering the larynx, he should be directed to take nothing but thick liquids. A little arrow-root may be used for giving a proper consistence to the fluids. By thickening the drink (in the way directed) it will be much less likely to pass, beneath the edges of the epiglottis, into the larynx. It is also well to direct the patient to take the drink at a draught—to gulp it down, so to speak—not to sip it. This mode of procedure makes the act of deglutition continuous instead of intermittent, and under these circumstances the passage of food into the larynx is much less likely to occur.

Preventive treatment is the only plan which can be adopted with satisfactory results: congestion of the larynx, therefore, in phthisical persons must be treated with the greatest diligence. The most proper local treatment should be adopted; complete rest of the vocal organ enforced; and, above all, suitable atmospheric conditions, if possible, obtained. A warm, dry, and uniform temperature is the grand desideratum.

SYPHILIS.

The laryngeal phenomena of syphilis differ at different epochs of the constitutional disease, and must therefore be considered separately. In *secondary syphilis*, condylomata are the most characterised condition, but chronic hyperæmia (without the mucous tubercles) and superficial ulceration are often met with. Condylomata, occurring in the larynx, present a similar appearance to those found in the pharynx and elsewhere; that is to say, they are raised patches of the mucous membrane.

An elaborate article has been published by Gerhardt and Roth [1] on the subject, and by these observers they are described as being papillary formations, uneven, whitish, smooth or jagged prominences, variously situated in the larynx and of various size and extent. These morbid projections were found most frequently on the vocal cords, on the inter-arytenoid fold, and in those situations which by friction become mechanically irritated. Gerhardt found these condylomata present in 20 per cent. of the patients suffering from secondary syphilis. This proportion, however, has not been found by other observers. In fifty-two cases of well-marked secondary syphilis, which I was kindly permitted to examine at the Lock Hospital in the year 1863, con-dylomata were only found in two cases, that is to say in less than 4 per cent. Gerhardt's cases, forty-four in number, were in the Venereal Department of a General Hospital, and therefore may well be compared with those at the Lock Hospital. The difference is very remarkable. At the Hospital for Diseases of the Throat we constantly meet with con-dylomata of the larynx, but the proportionate frequency of laryngeal con-dylomata in the constitutional complaint of course cannot be ascertained at that institution. The inter-arytenoid commissure and the epiglottis are the parts which I have most frequently observed to be affected. In addition to the condylomata of secondary syphilis, superficial ulcerations of a limited extent are also occasionally met with ; there is also sometimes very obstinate congestion of the mucous membrane, but it is impossible to tell whether the latter condition is due to the syphilitic dyscrasia. As regards the treatment, there is little to be said ; the condylomata rapidly disappear under local treatment of a stimulating character, and probably often spontaneously. In the cases reported by Gerhardt this condition was removed by a mercurial course ; the superficial ulcerations may be cured by the common astringent solutions.

In *tertiary syphilis,* rapid, deep, and extensive ulceration is the cha-racteristic morbid condition of the larynx. The ulcerative process frequently destroys the mucous and sub-mucous tissues to a very con-siderable extent, and the muscles and perichondrium are sometimes attacked.[2] The ulcerative process is often associated with an œdema-tous tendency ; in the latter case, the laryngeal œdema seems often to occur as an extension of disease from the pharynx. Even when the ulcerative process is arrested, however, the danger does not cease ; for the cicatrices often undergo a degree of contraction which greatly interferes with the calibre of the larynx. Numerous cases of this sort have come under my notice, and there are many pathological speci-mens which illustrate it.[3] The epiglottis is peculiarly prone to be affected by syphilitic ulceration. Whilst ulceration is attacking the epiglottis, great dysphagia is generally experienced ; but when the ulcers are healed, swallowing can generally be effected without trouble,

[1] Virchow, Archiv, Bd. xxxi. 1861, Hft. 1, § 7.
[2] Specimen No. 38, W. Series, St. Thomas's Hospital.
[3] Guy's Hosp. Mus. No. 1655-90, and St. Thomas's Hosp. Mus. No. 22, W Series.

even though nearly the whole valve is destroyed. When the walls of the pharynx are also ulcerated, there is danger of the edges of the epiglottis uniting with the pharynx. This condition gives rise to one of the most serious forms of dysphagia.

In these advanced stages syphilitic gummata are sometimes formed, not only in the tongue and pharynx, but in the muscles and sub-mucous tissues of the larynx. These generally soften and ulcerate. The later forms of syphilitic ulcerations should be treated consti-tutionally with iodide of potassium. Five, ten, or in some cases twenty grains may be given with advantage, in combination with ammonia. By largely diluting the medicine with water, its effect is increased, and it does not irritate the throat in being swallowed. The ulcerated surface should be touched every day with the solid nitrate of silver. For this purpose a piece of aluminium wire, suitably curved, and coated with fused nitrate of silver, should be used.

The ulcerative process, though of the most active character, is almost always very tractable under this treatment; in no stage of the disease does it appear to me to be necessary or desirable to use mercury. The chronic laryngitis sometimes met with in syphilitic persons (associated as it generally is with chronic bronchitis) resists every kind of treatment.

SECONDARY ŒDEMA.

Œdema may occur as a sequel of Bright's disease, and possibly as the result of cardiac or venous obstruction. Dr. Fauvel has applied the term "*aphonie albuminurique*" to the laryngeal œdema occasionally met with in renal disease, but Dr. George Johnson—an acknowledged authority on diseases of the kidney, and an accomplished laryngo-scopist—is of opinion that "Dr. Fauvel has considerably over-estimated the frequency and importance of œdema of the larynx as a result of Bright's disease."[1] Though I have seen a great number of cases of laryngeal œdema, I have never met with it as a complication of renal disease, but that it may occur is shown by the history of a specimen[2] in Guy's Hospital, and by the report of cases under the care of Dr. Rees and Dr. Barlow. Œdema is often the consequence of necrosis of the cartilages, and has been referred to under the disease (Laryngeal Phthisis) in which that morbid process most frequently takes place. It also sometimes occurs, as already shown, in the exanthemata : here it is more probably the result of low inflammatory action than of simple dynamic causes.

The treatment should be the same as that recommended for acute laryngitis.

[1] The Laryngoscope, 1864.
[2] No. 179,650. Lancet, Sept. 5, 1863, vol. ii. p. 277, and vol. i. Feb. 27, 1864.

APPENDIX, ON THE USE OF THE LARYNGOSCOPE.

This instrument, constructed for obtaining a view of the interior of the larynx during life, consists of two parts—(1) a small mirror fixed to a long slender shank, which is introduced to the back of the throat; and (2) an apparatus for throwing a strong light (solar or artificial) on to the small mirror. For this purpose either (*a*) a second (larger) mirror, which reflects the light from a lamp or the solar rays on to the throat-mirror, may be used; or (*b*) the luminous rays from a lamp may be collected and thrown directly on to the smaller mirror, by means of a lens placed in front of the flame. The former method is called "illumination by reflection;" the latter, "direct illumination."

HISTORY.—Various independent attempts to examine the larynx have been made at different times by different practitioners. Levret, a distinguished French physician, as far as bibliographical research at present goes, seems to have been the first to invent a laryngeal mirror. This occurred in the year 1743. In the beginning of the present century, Bozzini contrived a Laryngoscope, which to a certain extent complied with the conditions contained in the above definition; but being clumsily constructed, it could not be used effectively. In the year 1825, an unsuccessful attempt to inspect the glottis was made by M. Cagniard de Latour; and a few years later, in 1829, Dr. Benjamin Guy Babington exhibited, at the Hunterian Society of London, a Laryngoscope which, excepting that a hand mirror was used instead of a concave circular reflector attached to the operator's head, closely resembled the modern instrument. In later times,[1] Senn, Bennati, Baumès, Liston, Warden, and Avery made attempts or suggestions towards obtaining a view of the larynx during life; but it was left for M. Garcia to lay the foundation of a method of examination, which, through the genius and perseverance of Professor Czermak, at once reached a high degree of perfection. The employment of the Laryngoscope in practical medicine dates from a paper published by Czermak in 1858.[2]

The Laryngeal Mirror.—The throat-mirror should be of glass backed with a coating of silver (not amalgam, as this is much more readily damaged by heat), mounted in German silver, and fixed at an angle of about 120° to a slender shank or rod about four inches in

For further historical details see the author's treatise "On the Use of the Laryngoscope," chap. i. 3d Edition. (Longman and Co.)
Wien Medizin. Wochenschrift.

length of the same material. The shank of the mirror is fixed into
a hollow wooden or ivory handle, about three inches in length and
a quarter of an inch in thickness. A laryngeal mirror, the reflecting
surface of which is about four-fifths of an inch in diameter, will be
found convenient in most cases; where the distance between the
uvula and posterior wall of the pharynx, however, is great, the largest
size mirror, about one inch in diameter, answers best; in the case of
children, a mirror about half an inch in diameter should be used.
Circular mirrors cause the least inconvenience, but where the tonsils
are very large, oval or ovoid mirrors can be most easily employed.

Illumination by Reflection.—For throwing a strong light on to the
laryngeal mirror, and thus into the larynx, it will be found most
convenient to employ a circular and slightly concave mirror about
three inches and a half in diameter, and having a focal distance of
about twelve or fourteen inches. When the solar rays are reflected
into the throat, the surface of the mirror should be plane. The mirror
should be attached in some way to the operator's head, and may be
worn either opposite one of the eyes (Czermak), in front of the nose
and mouth (Bruns), or on the forehead (Johnson, Fournié). I follow
Czermak's plan. The reflector may be attached to the operator's
head, either by a spectacle-frame (Semeleder)—and in this case the
upper half of the rim of the eye-piece of the spectacle-frame may be
conveniently removed—or by a frontal band (Kramer). The mirror
should be connected with its support by a ball-and-socket joint. In
making an examination after the manner of Czermak, the reflector
should be perforated by an oblong hole, the long diameter of which
should correspond with the long diameter of the eye.

Any lamp that gives a bright steady light answers the purpose
perfectly well. A moderator, paraffin, or argand gas-burner, will each
be found convenient. My " Rack-movement [1] Lamp " is perhaps the
most convenient illuminating apparatus that exists. It is now
employed at most of the London hospitals, and is very suitable for
the private consulting room. For strengthening the light a lens may
be employed, and various lamps and lanterns have been contrived
for the purpose. The "light concentrator," which forms a part of my
rack-movement lamp, will be found useful in Laryngoscopy, for,
whilst enclosing the lateral rays, it collects all those which can
possibly be conveyed to the reflector.

Direct Illumination.—The best mode of using direct light is that
employed by most of the French laryngoscopists. The lamp, pro-
vided with a lens on the side facing the patient, is placed on a table
about a foot wide and three feet long. The observer sits on one
side of the table, and facing him, on the other side, is the patient.
The lamp, provided with a strong lens on the side of the patient, and
screened towards the practitioner, is placed on the table between
them. In operating, the practitioner has one arm round each side of
the lamp. The method employed by Stoerk and Walker, in which

[1] Made by Mayer and Meltzer, 59, Great Portland Street.

direct light (strengthened by a glass globe of water acting as a lens) is thrown on to the laryngeal mirror, is less perfect on account of the lateral deflexion which the rays undergo after impinging on the laryngeal mirror. For demonstrating to a class, the oxy-hydrogen light,[1] as employed at the Hospital for Diseases of the Throat, is the most perfect arrangement.

Method of Examination.—The patient should sit upright, facing the observer, with his head inclined very slightly backwards. The observer's eyes should be about one foot distant from the patient's mouth, and a lamp burning with a strong clear light should be placed on a table at the side of the patient, the flame of the lamp being on a level with the patient's eyes. The observer should now put on the spectacle-frame with the reflector attached, and, directing the patient to open his mouth, should endeavour to throw a disk of light on to the fauces, so that the centre of the disk corresponds with the base of the uvula. When the observer has gained dexterity in throwing the light, the patient should be directed to open his mouth widely, and to put out his tongue; and the operator should hold the protruded organ between the finger and thumb of his left hand, previously enveloped in a soft cloth or towel. In thus keeping the tongue out, the greatest gentleness should be used, as the employment of force, by exciting reflex action, only defeats the object in view. Holding the laryngeal mirror, previously warmed over the lamp (to prevent the condensation of the breath on the surface), like a pen in the right hand, the operator should now introduce it to the back of the throat, its face being directed downwards and kept as far as possible from the tongue. The posterior surface of the mirror ought to rest slightly on the base of the uvula, which should be gently pushed rather upwards and backwards towards the posterior nares. The plane of the mirror should form an angle of about 45° with the horizon.

Where the tongue forms an arched prominence at the back of the mouth, the patient should be directed to inspire deeply, or to produce some vocal sound; these acts cause an elevation of the uvula, and thus facilitate the introduction of the mirror. It is better to introduce the mirror several times, and keep it *in situ* only a few seconds, than to allow it to remain in the mouth too long, and thereby produce an irritation which prevents further examination at the same sitting. When the epiglottis is large and-pendent, the mirror should be introduced lower in the fauces, and more perpendicularly than is usually suitable.

The Laryngeal Image.—In some cases, on introducing the laryngeal mirror, only the epiglottis may be visible, with perhaps just the tips of the capitula Santorini at the posterior part; whilst in others the ary-epiglottic folds, the ventricular bands, the vocal cords, the small cartilages above the glottis, the cricoid cartilage, the rings of the trachea (and, perhaps, even the bifurcation of that tube), can be seen

[1] For a description of the apparatus, see *Medical Times and Gazette*, July 24, 1869.

with perfect distinctness. The appearance of parts is shown in the annexed drawings :—

FIG. 1. — LARYNGOSCOPIC DRAWING, SHOWING THE VOCAL CORDS DRAWN WIDELY APART, AND THE POSITION OF THE VARIOUS PARTS ABOVE AND BELOW THE GLOTTIS, DURING QUIET INSPIRATION.

ge. glosso-epiglottic folds ; u. upper surface of epiglottis ; l. lip of epiglottis ; c. cushion of epiglottis ; v. ventricle of larynx ; ae. ary-epiglottic fold ; cW. cartilage of Wrisberg ; cS. capitulum Santorini ; com. arytenoid commissure ; vc. vocal cord ; vb. ventricular band ; pv. processus vocalis ; cc. cricoid cartilage ; t. rings of trachea.

FIG. 2.—LARYNGOSCOPIC DRAWING, SHOWING THE APPROXIMATION OF THE VOCAL CORDS, AND THE POSITION OF THE VARIOUS PARTS, IN THE ACT OF VOCALIZATION.

fi. fossa innominata ; hf. hyoid fossa ; ch. cornu of hyoid bone ; cW. cartilage of Wrisberg ; cS. capitulum Santorini ; a. arytenoid cartilages ; com. arytenoid commissure ; pv. processus vocalis.

But to properly understand their relation, this book should be held at the same inclination as that which the laryngeal mirror occupies when *in situ* (that is to say, at an angle of 45° with the horizon, the foot of the page being furthest from the observer). The only inversion which takes place in the formation of the image is in the antero-posterior direction ; the part which in reality is nearest to the observer, the anterior insertion of the vocal cords, becoming furthest in the image, and the posterior commissure, which in reality is farthest from the observer, becoming nearest in the image. With regard to the lateral and vertical relations of parts, no inversion takes place. That which is on the right side of the larynx (the right vocal cord, for instance), appears on the right side of the mirror, and that which is on the left side of the larynx, on the left side of the mirror ; in the same way the part which is highest in the larynx (the epiglottis) is highest in the mirror, and the parts lower down (the arytenoid cartilages) are at the lower part of the mirror. It is only when the image is transferred to paper, and becomes a drawing, that its symmetrical character can give rise to mistaken notions concerning inversion.

It is necessary to make a few remarks on the normal colour of the different parts. *The epiglottis* is of a dirty pinkish hue on the upper surface ; its lip (or free edge, and the immediately adjacent under surface) is of a decidedly yellow colour ; whilst the cushion (and rest of the under surface, when visible) is invariably bright red. The *ary-epiglottic folds* are about the same colour as the mucous membrane of the gums, the cartilages situated in them being of a somewhat deeper tint. *The ventricular bands* are of a bright colour, being of about the same shade as the mucous lining the lips. *The vocal cords* should be

pearly white, like the conjunctiva of a child. The cricoid cartilages and tracheal rings are of a yellow colour, and the mucous membrane between them bright red.

The Introduction of Instruments within the Larynx.—In applying local remedies to, or operating on, the larynx, by the aid of reflected light, as the right hand is required for the instrument used, the laryngeal mirror should be introduced with the left hand. In this case, the patient must hold his own tongue out.

Infra-glottic Laryngoscopy.—In some cases, after tracheotomy has been performed, and where a tube is worn, valuable evidence may be derived by introducing a minute mirror, with its face directed obliquely upwards, through the fenestrated canula. On account of the size of the mirror, it is necessarily made of steel; and as both its size and material cause it to cool very rapidly, a coating of glycerine will be found convenient for neutralizing the effects of the condensation of the water contained in the expired air.

§ III. Diseases of the Respiratory System (*continued*).

B. Diseases of the Thoracic Organs.

1. Emphysema.
2. Asthma.
3. Phthisis.
4. Cancer.
5. Acute Pneumonia.
6. Chronic Pneumonia.
7. Syphilitic Affections of the Lung.
8. Brown Induration of the Lung.
9. Cirrhosis.
10. Apneumatosis.
11. Bronchitis.
12. Pleurodynia.
13. Pleurisy.
14. Hydrothorax.

15. Pneumo-Thorax.

EMPHYSEMA OF THE LUNGS.

By Sir William Jenner, Bart., M.D. Lond., D.C.L. Oxon., F.R.S.

DEFINITION.—Relative excess of air in a part or the whole of the lungs.

The relative excess of air may be the result of increase in the quantity of air in the vesicles, of diminution in the solid tissues of the lung, or of the presence of air in lung structures which in health do not contain air.

Pulmonary Emphysema may be divided into—

1. Interlobular, extra-vesicular, or extra-alveolar Emphysema.

2. Vesicular or alveolar Emphysema.

This division, first made by Laennec, has been adopted by all subsequent writers on the subject.

INTERLOBULAR, EXTRA-VESICULAR, OR EXTRA-ALVEOLAR EMPHYSEMA.

DEFINITION.—Air in the connective tissue of the lung.

The connective tissue of the lung is seated chiefly, at least, between the lobules and under the pleura. The air in extra-vesicular Emphysema occupies the meshes of this connective tissue.

When air is present in the connective tissue between the lobules, it accumulates in small bubbles of tolerably equal size, separated from each other by bands of tissue, so that the surface of the lung looks as if streaked or crossed by rows of small beads. When air is in the sub-pleural tissue it forms air-blebs, sometimes of very large size.

Air may be formed after death in the connective tissue of the lungs by decomposition; it may be generated there during life by gangrene; and it may be extravasated into the same tissue in consequence of rupture of the normally air-containing structures of the lung. When formed by decomposition after death, the gas is usually seated in the interlobular tissue; when generated by gangrene, in the sub-pleural tissue; and when extravasated from the air-vesicles, it commonly occupies both situations.

Air extravasated into the connective tissue of the lung occasionally finds its way into the posterior mediastinum, and thence into the subcutaneous tissue of the neck, face, trunk, &c.

Rupture of the normal air-vesicles may be the result of injury

inflicted from without, or of the pressure of the air on their inner surface during violent expiratory efforts made when the glottis is closed, *e.g.*, during cough and parturient efforts. The distension of the air-vesicles by inspiratory efforts is never great enough to cause their rupture.

Interlobular Emphysema is a condition of little importance. When the air finds its way through the connective tissue of the posterior mediastinum into the subcutaneous tissue, the air is quickly absorbed, and in a few days no trace of the Emphysema is to be detected.

With the exception of the cases in which the air reaches the subcutaneous tissue, the diagnosis of pulmonary extra-vesicular Emphysema is impossible, and even in these cases there are no pulmonary signs or symptoms to indicate the existence of the local lesion.

Should the existence of extra-vesicular Emphysema be ascertained, no treatment is needed.

PULMONARY VESICULAR EMPHYSEMA.

DEFINITION.—Increase in the capacity and size of the air-vesicles of the lungs.

Pulmonary Vesicular Emphysema is a very common, and frequently a grave disease.

Causes of increase in the capacity and size of the air-vesicles.— All forms and degrees of Pulmonary Vesicular Emphysema have their origin either in destruction of the partitions between the air-vesicles, or in over-distension of individual air-vesicles. In the former case, two or more air-vesicles are, by the primary lesion, thrown into one ; in the latter, each air-vesicle is, by an over-distending force, increased in capacity and size.

It is improbable that nutritive changes in the tissue of the walls of any hollow viscus ever lead directly to expansion of its cavity. But changes in the walls of a hollow viscus may weaken their resisting power and so favour the expansion of its cavity ; and again, changes in the walls of a hollow viscus may cause a dilatation to be permanent, which otherwise would have been temporary.

Changes in the walls of a hollow viscus, which strengthen their resisting power, may, at the same time, weaken their contractile power. Walls so changed may resist a dilating force longer than healthy walls, but should the dilating force be sufficient to stretch them, the dilatation of the cavity they enclose is permanent. The walls are indeed stronger, but then the cavity is more likely to suffer *permanent* dilatation.

The causes of increase in the capacity and size of the air-vesicles of the lungs are then divisible into :—

1. The forces which determine their over-distension ;
2. The conditions which favour their over-distension ;
3. The conditions which render their over-distension permanent;

4. The lesions of structure by which two or more vesicles are thrown into one.

Although this division should always be kept in view when considering the causes of Pulmonary Vesicular Emphysema, it will be better, in an article such as this, to consider the causes included in the second and fourth divisions incidentally, as occasion arises, when treating of the causes included in the first and third divisions.

Determining causes of over-distension of the air-vesicles.—Pressure of air on the inside of the air-vesicles is the force which directly causes their normal expansion; increase in that pressure is the immediate cause of their over-distension.

Excess of pressure of air on the inside of the pulmonary air-vesicles (of the whole or of a part of the lung) may be brought to bear,

(*a*) By excessive expansion of the chest-walls;

(*b*) By normal expansion of the chest-walls, when disseminated portions of the lung are shrunken, and no longer admit air;

(*c*) By unequal compression of the lung at the moment when there is impediment to the free escape of air from its air-containing parts.

(*a*) In health, inspiration is effected by muscular effort, ordinary expiration chiefly by the elasticity of the thoracic parietes and of the lung textures. The muscular effort of inspiration overcomes the resistance to the entrance of the air into the air-vesicles, offered by the elasticity of the lungs and of the walls of the thorax; the muscular effort ceasing, the elasticity of these parts is sufficient to accomplish the ordinary expiratory act.

The elasticity of the ribs and of their cartilages diminishes considerably as age advances, while in a large number of cases the muscles of inspiration continue as powerful as, and are sometimes more powerful than, in early life. The result of inspiratory expansion of the chest being constantly accomplished by the action of muscles undiminished in power and activity, and of expiratory diminution of the chest being constantly performed incompletely and imperfectly by thoracic parietes the elasticity of which is diminished, is gradual expansion of the chest-walls, increased capacity of the chest, and dilatation of the air-vesicles of the lungs. The capacity of the chest not being reduced to its normal size during expiration, the inspiratory effort is made on a chest retaining too much air in the lung-vesicles, and thus, especially if there be repeated and powerful calls on the inspiratory power, as from cough or great muscular effort, the result is considerable over-distension of the air-vesicles.[1]

In the same way lessened elasticity of the lungs from age-degeneration, or other cause, without loss of power in the muscles of respiration, leads to increase in the capacity of the thorax, and over-distension of the air-vesicles. The excessive expansion of the thorax, and therefore the dilatation of the air-vesicles in this, as in the last

[1] Dr. G. Budd, in a paper on Pulmonary Vesicular Emphysema, published in the Med.-Chir. Soc. Trans. for 1840, clearly pointed out the part which loss of elasticity of the lung plays in the production of emphysema.

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PULMONARY VESICULAR EMPHYSEMA.

DEFINITION.—Increase in the capacity and size of the air-vesicles of the lungs.

Pulmonary Vesicular Emphysema is a very common, and frequently a grave disease.

Causes of increase in the capacity and size of the air-vesicles.— All forms and degrees of Pulmonary Vesicular Emphysema have their origin either in destruction of the partitions between the air-vesicles, or in over-distension of individual air-vesicles. In the former case, two or more air-vesicles are, by the primary lesion, thrown into one; in the latter, each air-vesicle is, by an over-distending force, increased in capacity and size.

It is improbable that nutritive changes in the tissue of the walls of any hollow viscus ever lead directly to expansion of its cavity. But changes in the walls of a hollow viscus may weaken their resisting power and so favour the expansion of its cavity; and again, changes in the walls of a hollow viscus may cause a dilatation to be permanent, which otherwise would have been temporary.

Changes in the walls of a hollow viscus, which strengthen their resisting power, may, at the same time, weaken their contractile power. Walls so changed may resist a dilating force longer than healthy walls, but should the dilating force be sufficient to stretch them, the dilatation of the cavity they enclose is permanent. The walls are indeed stronger, but then the cavity is more likely to suffer *permanent* dilatation.

The causes of increase in the capacity and size of the air-vesicles of the lungs are then divisible into :—

1. The forces which determine their over-distension ;
2. The conditions which favour their over-distension ;
3. The conditions which render their over-distension permanent;

4. The lesions of structure by which two or more vesicles are thrown into one.

Although this division should always be kept in view when considering the causes of Pulmonary Vesicular Emphysema, it will be better, in an article such as this, to consider the causes included in the second and fourth divisions incidentally, as occasion arises, when treating of the causes included in the first and third divisions.

Determining causes of over-distension of the air-vesicles.—Pressure of air on the inside of the air-vesicles is the force which directly causes their normal expansion; increase in that pressure is the immediate cause of their over-distension.

Excess of pressure of air on the inside of the pulmonary air-vesicles (of the whole or of a part of the lung) may be brought to bear,

(*a*) By excessive expansion of the chest-walls;

(*b*) By normal expansion of the chest-walls, when disseminated portions of the lung are shrunken, and no longer admit air;

(*c*) By unequal compression of the lung at the moment when there is impediment to the free escape of air from its air-containing parts.

(*a*) In health, inspiration is effected by muscular effort, ordinary expiration chiefly by the elasticity of the thoracic parietes and of the lung textures. The muscular effort of inspiration overcomes the resistance to the entrance of the air into the air-vesicles, offered by the elasticity of the lungs and of the walls of the thorax; the muscular effort ceasing, the elasticity of these parts is sufficient to accomplish the ordinary expiratory act.

The elasticity of the ribs and of their cartilages diminishes considerably as age advances, while in a large number of cases the muscles of inspiration continue as powerful as, and are sometimes more powerful than, in early life. The result of inspiratory expansion of the chest being constantly accomplished by the action of muscles undiminished in power and activity, and of expiratory diminution of the chest being constantly performed incompletely and imperfectly by thoracic parietes the elasticity of which is diminished, is gradual expansion of the chest-walls, increased capacity of the chest, and dilatation of the air-vesicles of the lungs. The capacity of the chest not being reduced to its normal size during expiration, the inspiratory effort is made on a chest retaining too much air in the lung-vesicles, and thus, especially if there be repeated and powerful calls on the inspiratory power, as from cough or great muscular effort, the result is considerable over-distension of the air-vesicles.[1]

In the same way lessened elasticity of the lungs from age-degeneration, or other cause, without loss of power in the muscles of respiration, leads to increase in the capacity of the thorax, and over-distension of the air-vesicles. The excessive expansion of the thorax, and therefore the dilatation of the air-vesicles in this, as in the last

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[1] Dr. G. Budd, in a paper on Pulmonary Vesicular Emphysema, published in the Med.-Chir. Soc. Trans. for 1840, clearly pointed out the part which loss of elasticity of the lung plays in the production of emphysema.

case, is determined by extreme muscular inspiratory action—the necessary result of deficient ordinary expiratory power.[1]

Another cause of increased expansion of the thorax has been described by Freund. He says that persons of all ages, from twenty years upwards, the well-nourished as much as the withered and decrepit, are liable to a chronic disease of the cartilages of the ribs, which results in their hypertrophy and increased firmness and rigidity, and in diminution of their elasticity. As this increase in the size of the cartilages takes place in all directions, the ribs and sternum are separated from each other more than they are in health; the ribs being forced outwards and upwards, and the sternum forwards and a little upwards. The capacity of the thorax is thus (Freund says) increased, and the lungs proportionately over-distended. It has been contended by later writers that Freund exaggerated the frequency at least of this affection; that he supposed changes in the cartilages resulting from their stretching to be the primary affection—in fact, that he took the *effects* of the action of the determining cause for the determining cause itself. For the capacity of the thorax to be increased under the conditions named by Freund, the diaphragm must continue to be at the termination of ordinary expiration on as low a level as in health. Usually, however—and perhaps always when the cartilages lengthen—they bend so as to form an angle, with its concavity upwards, about their centre.

(*b*) It is evident that if disseminated portions of lung are from any pathological condition diminished in size and no longer admit air, and that if, at the same time, the chest-walls expand during inspiration to the same amount as in health, the air-admitting vesicles must be over-distended in proportion to the number of cells into which no air enters, and the degree to which the airless vesicles are diminished in size. Thus, in certain cases of bronchitis, disseminated lobular collapse is common. The collapsed lobules are smaller in bulk than are the air-containing lobules—their vesicles admit no air during inspiration. The necessary result is, that if the chest-walls expand to the same degree as before the establishment of collapse, and so inspire an equal quantity of air, the capacity of all the air-vesicles still pervious must be increased.[2]

(*c*) If a lung removed from the body be moderately inflated, and the bronchus leading to it be tied, and then the substance of the organ be compressed at one part, over-distension of the air-vesicles of the uncompressed part is produced. Should the compressed part be large, and the compression considerable, even rupture of the air-vesicles of the uncompressed part may result. Under the conditions supposed, air is forced from the compressed parts of the lung into the air-admitting structures of the uncompressed parts of the lung.[3]

[1] See p. 482.   [2] This point has been excellently well brought out by Dr. W. Gairdner.
[3] This expiratory theory was first advanced by Mendelssohn, in a very able paper, "Der Mechanismus der Respiration und Circulation," 1845. The writer of this article was unacquainted with Mendelssohn's paper when he advanced the same theory in 1857, and so far as he knows the existence of Mendelssohn's paper was unknown in this country, and rarely *if ever* referred to abroad till Biemer's article appeared in 1867.

The conditions essential to the over-distension of the air-vesicles here present are :—

(*a*) Inflation of the lung;

(*b*) Closure of the natural passage for the escape of air from the lung.

(*c*) Unequal pressure on the lung;

(*d*) Unequal support of different parts of the lung.

During violent cough and great muscular effort these essential conditions are fulfilled :—

(*a*) Preparatory to cough and to great muscular effort, a deep inspiration is taken, *i.e.*, the lungs are inflated.

(*b*) Then the glottis is closed, *i.e.*, the air is prevented escaping by the natural channel.

(*c*) Then, by the action of the expiratory muscles, the lungs are strongly compressed, and an examination of the structure of the thoracic walls at once shows that the compression must be unequal.

(*d*) Examination of the structure of the walls of the chest also shows that the support offered to the lungs by those walls is very different in degree at different parts.[1]

Again, when blowing a wind instrument the chest is expanded to its utmost, and then the chest-walls compress the inflated lungs— the air cannot escape as freely through the instrument as through the open glottis, and the mechanical effect is over-distension of the air-cells of the least compressed and least supported parts in proportion to the impediment to the escape of air and the force with which it is attempted to drive the air forward.

The over-distension of the air-cells thus effected will be in proportion to the amount of inflation of the whole lung, to the firmness with which the glottis is closed, or the smallness of the aperture of the wind instrument, or other obstacle to the free escape of air;[2] to the extent, degree, and difference in the force of compression exercised on the several parts of the lung at the same moment; and to the deficiency of support afforded to the less compressed parts by the thoracic parietes. The greater and the more extensive the compression of one part of the lung, and the less the compression of the other, the greater will be the distension of the air-cells in the less compressed part; and the less the imperfectly compressed parts are supported by the thoracic parietes, the greater will be the distension of their air-vesicles.

It would at first sight appear that the over-distension of the vesicles should be in all cases limited to the less compressed and the less supported parts of the lungs, but on further examination it will be seen that this opinion is erroneous. Thus, if from some change in the walls of the chest or of the air-vesicles, the latter continue over-distended after the force which directly determined

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[1] For details on this point see the author's paper on the determining causes of Pulmonary Vesicular Emphysema, in the Medico-Chirurg. Soc. Trans. 1857.

[2] Dr. Budd's case.

their over-distension has ceased to act, or in other words, if there be permanent dilatation of the air-cells, then the size of the chest and of the lungs is permanently increased.

The portions of lung corresponding to the intercostal spaces are less compressed and less supported just before violent expiration than are the parts immediately under the ribs themselves. Now with every increase in the size of the lungs, or thorax, or both, the relative positions of the lungs and ribs are changed. As the chest enlarges, the ribs assume a more horizontal position, the lower intercostal spaces become wider, and their supporting power by so much diminished.

By these changes in the lungs and in the chest-walls their relative positions are being constantly shifted, and fresh portions of the lungs are being constantly brought to correspond to the ribs and to the intercostal spaces, &c., and thus, ultimately, the air-vesicles of the whole lung may be over-distended. But when the air-vesicles of the whole lung are thus over-distended, the dilatation of the vesicles at the apex and margin of the lung is in excess of the dilatation of the vesicles of other parts. Strong expiratory effort, while there is impediment to the free escape of air from a part or whole of the lung, is now admitted to be the most common efficient determining cause of over-distension of the air-vesicles.

Pulmonary Vesicular Emphysema is very common in horses, and for this reason, viz., that they are constantly making powerful muscular efforts with closed glottis. No one who watches a horse draw a heavy load up a short steep incline on a damp cold day can doubt this. While making the effort, the horse holds its breath, having previously inflated its lungs—no sooner, however, does the animal cease its effort than the glottis is opened and the air suddenly expressed from the lungs. The degree to which the air was compressed during the powerful effort (and the consequent strain on the less compressed and less supported part of the lung) may be judged by the distance to which, and the sudden violence with which, the cloud of breath-vapours is seen to be driven forth.

Permanence-securing causes, or the conditions which render over-distension, or increase in the capacity and size of the air-vesicles of the lungs permanent.—Whatever destroys the partitions between adjacent air-vesicles, and whatever permanently diminishes the ordinary or habitual respiratory power, must, to a like degree, be a permanence-securing cause of increase in the capacity and size of the air-vesicles. The permanence-securing causes, therefore, are :—

1. Direct injury to the elasticity of the walls of the air-vesicles;

2. Permanent diminution of the power of supporting or compressing the lung, at any one part, during violent expiratory efforts;

3. Changes in the structure of the parietes of the thorax, which permanently diminish their elasticity, and therefore their ordinary or habitual expiratory power;

4. Chronic changes in the structure of the lungs, which permanently diminish their elasticity, and therefore their expiratory power;

5. Atrophy of the septa between the air-vesicles of the lungs, by which two or more vesicles are thrown into one.

1. If the forces which expel the air from the air-vesicles, viz., the elasticity of the thoracic parietes, and the elasticity of the walls of the air-vesicles, are at the termination of over-distension of the vesicles in a state of health, then the force determining their over-distension ceasing to act, the air-vesicles return to their natural size; but if, as very rarely happens, the air-vesicles have been *very* greatly over-distended, or kept for a very long time over-distended, or have been very repeatedly over-distended, then the elasticity of the walls of the air-vesicles may be permanently injured, and the over-distending force ceasing to act, they do not recover their normal dimensions. They are under the circumstances supposed permanently over-distended. The elastic structures of their walls have been directly injured by the over-distending force. So great even may be the force by which their over-distension has been effected, that the partitions between adjacent vesicles may be destroyed, and two or more vesicles thrown into one; or even, as has been previously mentioned, the destruction may have reached further, and air have been extravasated into the interlobular tissue.

2. The observations of Ziemssen on a case in which there was loss of muscular power in the four upper intercostal spaces, proves that this loss may be a cause of Vesicular Emphysema. In Ziemssen's case, during violent expiratory effort, these intercostal spaces no longer affording their normal support to the lung were forced outward so much as to stand above the level of the ribs. When the muscles of either intercostal spaces were stimulated to contract by Faradization, then the bulging during expiratory efforts of that intercostal space ceased, thus proving that want of muscular contraction at any part during expiratory effort, is a cause of over-distension of the air-vesicles of the lung at that point; and if the want of support be permanent, then certainly the over-distension will be permanent.

3. The degenerations of the ribs and cartilages incident to age, diminish their elasticity, and consequently diminish the expiratory power of the chest-walls. If, as was previously pointed out, the inspiratory muscles act perfectly when the expiratory force resulting from the resilience of the ribs and cartilages is diminished, dilatation of the thorax, over-distension of the air-vesicles, and enlargement of the lungs are determined.

As age-degeneration is a permanent lesion, the loss of elasticity resulting from it is permanent; the dilatation of the thorax, over-distension of the air-vesicles, and the enlargement of the lungs, is permanent. Age-degeneration of the ribs and their cartilages is, with perfectly acting inspiratory muscles, therefore a permanence-securing cause of Large-lunged Vesicular Emphysema.

The disease of the cartilages of the ribs described by Freund, once established, is permanent, and therefore, the over-distension of the air-vesicles due to the expansion of the chest resulting from it, is also permanent.

4, 5. Whatever changes in the lungs diminish their elasticity, to the same degree render permanent the over-distension of the air-vesicles determined by any of the forces previously enumerated.

Diminished elasticity of the lung may be the consequence of those changes in texture which result from repeated or long-continued congestion. After a part has been the seat of long-continued or of repeated congestion, it is, if an organ, indurated and toughened; if a tissue, toughened and thickened. If death occur long after the outset of the congestion, then a certain amount of wasting of the original structures is found to have taken place. In some cases, certainly, these changes are due to the formation among the normal anatomical elements of the part, of imperfectly developed connective, fibrous, or fibro-cellular tissue.

All degenerations of texture incident to age, are attended by more or less loss of elasticity.[1]

The degenerations incident to age,[2] as they affect the lung, may be divided thus :—

(a) Atrophy, or waste of all the anatomical constituents of the lung, with general diminution in its size. As the partitions between the air-vesicles atrophy, two or more vesicles are thrown into one. This form of atrophy has been supposed to be preceded by fatty degeneration.

(b) Thickening of the fibrous element of the lung, with more or less waste of some of its anatomical constituents. When the subject of this form of degeneration the size of the lung is often increased, and it may be considerably so.

In this latter form of age-degeneration there is, at the outset at least, no atrophy of the inspiratory muscles; while in the former, the muscles on the outside of the chest are wasted and pale, and the diaphragm is thin, lax, and in folds. In both, the ribs and cartilages are the seat of degenerative changes attended by loss of elasticity.

So, also, when the ribs and cartilages lose elasticity from age-degeneration, the lungs rarely preserve their normal elasticity; they too, commonly, like the ribs and cartilages, are suffering from age-degeneration.

[1] Diminution of elasticity is one of the most marked effects of the changes in nutrition incident to advancing age, e. g., of the skin, giving the aged look ; of the arteries, causing them to become tortuous or S shaped, at first when the part in which they are placed is shortened, as in flexion of the limbs, and then permanently ; of the intervertebral cartilages, of the elastic structures in the sole of the foot, the joints, the bones, &c.

[2] Those changes of nutrition which are the characteristics of age, and in fact constitute ld age, may occur, generally or locally, at an unusually early age. Thus, one man grows prematurely old as regards his jaws, another as regards his hair, another as regards the heart, &c.

The conjunction of diminished elasticity of the lungs and of the parietes, reduces the ordinary or habitually employed expiratory force to a minimum. Now, this being the case, if the muscles of inspiration and of expiration retain their normal power, then frequent cough, habitual straining at stool, moving heavy weights, climbing hills, blowing wind instruments, or other causes of repeated and powerful inspiratory efforts, followed by violent expiratory compression of the inflated lungs, with impediment to the escape of air, will be followed by great and permanent increase in the size of the thorax, and corresponding over-distension of the air-vesicles.

Changes in the lung, attended by loss of elasticity, said to be independent of age and of congestion, have been described by various authors.

M. Villemin thinks that the true anatomical structure of the walls of the air-vesicles is a network of capillary vessels, with a nucleus filling each intercapillary mesh, and elastic fibres on the inside of the vesicles crossing over the capillaries and intercapillary nuclei. "In Pulmonary Vesicular Emphysema," M. Villemin says, "the nuclei in the meshes of the capillary network hypertrophy, compression and atrophy of the capillaries follow; then the enlarged nuclei undergo fatty degeneration; they fall from their places in the walls of the air-vesicles, destruction of the elastic tissue and of more capillaries occurs; apertures are formed between adjacent vesicles, and finally, two or more vesicles are thrown into one."

"There is then," M. Villemin says, "a first stage of Emphysema, a true hypertrophy of the elements of the vesicular membrane; from this there naturally results an extension of that membrane, and an increase in the capacity of the vesicles."

It does not, however, necessarily follow, even though M. Villemin's anatomical observations be correct, that there is an increase in the size of the lung, as he supposes, because the walls of the air-vesicles are lengthened; for they might, under such circumstances, be folded on themselves. Moreover, the accuracy of these observations has been doubted. The so-called intercapillary nuclei are said by some observers to be epithelium on the inside of the air-vesicles.

"Changes in the nutrition of the lung," Freund says, "necessarily follow on the changed conditions of the respiratory movements due to the lengthening of the rib-cartilages, and these changes are attended by loss of elasticity and the other changes in the walls of the air-vesicles which follow on their continued over-distension."

Dr. Waters, while admitting that his investigations do not enable him to say what is the nature of the degeneration which leads to Emphysema, and that his microscopical researches on this point have yielded no results, adds, "I do not entertain the slightest doubt that the disease in its severer forms is of a constitutional nature."

Varieties of Pulmonary Vesicular Emphysema.—As over-distension of the air-vesicles may occur in perfectly healthy lungs, and in lungs

the seat of any of those pathological changes which impair their elasticity, and as, moreover, the dilatation may affect the air-vesicles of the whole, or of a great part of the lung, or may be limited to the air-cells of a small part of the lung, Pulmonary Vesicular Emphysema has been divided into varieties.

The various forms of Pulmonary Vesicular Emphysema may be described under the four following heads:—

Acute Vesicular Emphysema.

Chronic Local Emphysema.

Large-lunged (or Hypertrophous) Emphysema.

Small-lunged (or Atrophous) Emphysema.

Although perfect and uncomplicated specimens of each variety are common, cases of Pulmonary Vesicular Emphysema are frequently seen in practice and in the dead-house, in which these several varieties are conjoined in the same lung, and, again, cases which cannot at the time when they come under observation be referred absolutely to the one or the other group. The reasons for this are manifest when the etiology and the pathology of the affection are considered.

ACUTE VESICULAR EMPHYSEMA.—By Acute Pulmonary Vesicular Emphysema is signified acute over-distension of previously healthy air-vesicles.

The part of the lung, the air-vesicles of which are over-distended, is puffed up, is paler than it should be; the vesicles themselves, seen through the pleura, are manifestly larger than natural. The pallor is due solely to the excess of air in the vesicles stretching their walls, and so separating the capillaries further than should be from each other. The meshes of the capillary network on the walls of the air-vesicles are widened. Acute Vesicular Emphysema may be produced by too much air being drawn into the over-distended air-vesicles by inspiratory effort; or by too much air being driven into the air-cells of parts of the lungs by extreme compression of other parts by expiratory efforts, while the escape of the air by the natural outlet is prevented or retarded, *e.g.*, by closed glottis, narrowing of the trachea or bronchi.

Both these forces conspire to determine the occurrence of acute over-distension of the air-vesicles in acute bronchitis. In that disease disseminated collapse, and the consequent diminished bulk of lung, and increased desire for breath, lead to violent inspiratory efforts and over-distension of the pervious air-vesicles; while the frequent and violent expiratory efforts with closed glottis (preparatory to cough), determine over-distension of the air-vesicles of the less compressed and less supported parts of the lung.

When the ribs are greatly softened, as in some cases of rickets, the anterior margin of the lungs is the seat of Acute Vesicular Emphysema. The over-distension of the air-vesicles is produced partly by the compression of the lung at a little distance from its margin by the recession during inspiration of the ribs at their junction with

their cartilages, but chiefly by the great advance of the sternum and rib-cartilages during inspiration, these parts being thrust forward by the impressing ribs.

In Acute Pulmonary Vesicular Emphysema, the rule is that the air-vesicles resume their normal size as soon as, or very soon after, the over-distending force ceases to act. The walls of the air-vesicles and the adjacent tissues being healthy, they contract to their normal dimensions.

In comparatively rare cases, the over-distension is so great, so long-continued, or so frequently repeated, that the over-stretched walls of air-vesicles are injured, their elasticity is impaired, and the air-vesicles continue permanently larger than they should be.

It is in this way that severe and prolonged hooping-cough in children appears to produce Chronic Pulmonary Vesicular Emphysema. The over-distension especially affects the air-vesicles of the apex and anterior margin of the lungs, the air being forced into those parts during the violent expiratory efforts which precede the cough.

Symptoms.—If widely spread, and extreme, Acute Pulmonary Vesicular Emphysema causes increased resonance of the chest; the symptoms due to the lesion are masked and altogether lost in those proper to the disease to which it is secondary.

It requires no special treatment.

CHRONIC LOCAL EMPHYSEMA is characterised by extreme permanent over-distension of a few vesicles. The large vesicles are formed by the coalescence of several smaller. The largest may be as large as a poulet's egg, are not unfrequently the size of hazel-nuts, though more commonly not larger than peas. In the same group, vesicles are often found varying in size from a pin's head to a hazel-nut.

The walls of these large vesicles are never healthy; they are thick, opaque, wanting in elasticity, and vessels of some size frequently ramify on the larger. Threads composed of obliterated bronchi, the remains of vessels or of lung tissue, cross the cavity of the larger vesicles. Sometimes these vesicles communicate with a small bronchus; at others the bronchus leading to them is occluded.

The most common seat of Chronic Local Emphysema is the apex of the lung, then the anterior margin, and the margin of the base of the lung. At the apex, the Emphysema is often conjoined with the remains of old tubercle.

The pathology and mechanism of the production of Chronic Local Emphysema is best studied as it occurs at the apex of the lung, when that part is the seat of obsolescent or of calcified tubercle.

When tubercles obsolesce or calcify at the apex of the lung, a considerable portion of lung-tissue in their vicinity is usually the seat of chronic congestion and exudation of lymph. This portion of lung loses its porosity, becomes tough, inelastic, and puckered, *i.e.*, irregularly contracted. Here and there, however, portions of the lung-textures are damaged, not destroyed, so that some air-vesicles still admit air.

In health the inspiratory and expiratory forces are at a minimum at the apex. But during expiratory efforts with closed glottis, as in severe cough, the air is driven from the more compressed parts into the little compressed apex, and thus the vesicles still pervious to air are over-distended ; and, as their walls have, from the previous changes in the tissues of the apex of the lung, lost much of their elasticity, the over-distension is permanent. Every paroxysm of cough must add to their dilatation. The diminution in size of the apex assists, as a permissive cause, in the production of extreme Chronic Local Emphysema at the apex.

Thus in proportion to the loss in the elasticity of the air-admitting textures, to the frequency and the violence of the expiratory efforts with closed glottis, and to the permanent diminution in the size of the apex, will be the degree and the rapidity with which Local Emphysema at that part will be established.

The anterior margin of the lung, the margin of the base, the anterior inferior angle of the superior lobe of the left lung are, like the apex, very imperfectly compressed and supported during expira-tory efforts, and so air is forced powerfully into the vesicles of those parts ; and should the texture there be damaged at any time so as to diminish its elasticity, the result will be great dilatation of a few vesicles. The margins of the lungs are thus sometimes fringed with large vesicles.

Chronic Local Emphysema is always a secondary lesion. Its formation at the apex is the consequence, not the cause (as some have fancied) of the obsolescence of tubercles. Coincident with the obsolescence is damage to the air-admitting textures of the lung, and it is that damage which renders the Chronic Local Emphysema with large vesicles possible.

Symptoms.—The development of Emphysema at the apex of the lung, when that part is the seat of chronic consolidation with contraction, diminishes the depression of the shoulder, and of supra- and infra-clavicular regions, and increases the resonance of the same parts ; the dilated vesicles often projecting above and surrounding the solid textures. The dilatation of the vesicles may be so extensive and con-siderable as to cause supra-clavicular bulging either permanently or during cough.[1] It is unattended by other symptoms.

Treatment.—From the nature of the lesion, it will be understood that no treatment is required.

LARGE-LUNGED VESICULAR EMPHYSEMA.—By this name it is pro-posed to designate those cases in which there is over-distension of the air-vesicles of the whole, or of a large section of one or of both lungs, great increase in bulk of the lungs, or of the affected part of the lungs, and corresponding increase, local or general, in the capacity of

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[1] The bulging part is resonant and cannot, therefore, be confounded by a tolerably careful observer with the prominence of the same part due to distension of the veins during severe cough.

the thorax. The term Hypertrophous Pulmonary Vesicular Emphysema has been used to describe the same set of cases.[1]

General Large-lunged Vesicular Emphysema is a very serious disease. The symptoms directly due to it are grave; the diseased conditions dependent on it for their origin are very frequently fatal.

Thus a large proportion of cases of heart disease have their starting point in Large-lunged Vesicular Emphysema.

It rarely occurs in a marked form before the middle of life, and it more commonly affects those disposed to accumulation of fat in the subcutaneous tissue and internal parts. Lungs, the subject of this form of Vesicular Emphysema, are larger and drier than healthy lungs.[2]

The parts uncoloured by pigment are paler than healthy lung.

The lungs overlap the pericardium to a considerable extent, and meet above it even to near the top of the sternum; they have a down-cushion-like feel, and retain the impression of the fingers. When the thorax is opened they contract less than healthy lungs do under like circumstances.

Large-lunged Vesicular Emphysema is, in the great majority of cases, preceded by attacks of bronchitis, by congestion of the lungs, by dry winter cough, or by chronic bronchitis; that is to say, by diseases having as immediate consequences toughening and thickening of the tissues of the lung,[3] and severe cough; in other words, diminished elasticity of the lungs, and powerful expiratory efforts with closed glottis.

By far the most common determining cause then of the over-distension of the air-vesicles in Large-lunged Vesicular Emphysema is powerful expiratory effort with closed glottis; and the most common permanence-securing cause is the changes in the texture of the walls of the air-vesicles resulting from excess of blood in their capillaries.

[1] Large-lunged is by far the better of the two names, because it involves the expression of no opinion in regard of disputed facts. Many observers regard Pulmonary Vesicular Emphysema as atrophic from its outset—no matter how it originates. And it must be admitted that even when the disease has been hypertrophic when first established, the lungs may be greatly wasted in regard of their essential anatomical constituents before death. And again, in some cases of Large-lunged Vesicular Emphysema, as in those in which the occurrence of the disease is determined, and its continuance secured, by increase in the capacity of the chest from age-degeneration of the ribs and cartilages without diminution in the power of the respiratory muscles, the wasting and rarefaction may not be preceded by hypertrophy of any anatomical constituent of the lung.

[2] When Vesicular Emphysema follows on bronchitis, congestion of the lungs, and similar pathological conditions, the lungs, at the very outset of the disease, weigh more than in health, and would continue to do so were it not for the waste of the normal anatomical constituents of the lung—blood, blood-vessels—epithelium, or intercapillary nuclei —which follows on over-distension of the air-vesicles, and on the lesions which secure the permanence of their over-distension.

[3] To comprehend the relation between bronchitis, the changes following it in the walls of the air-vesicles, and the frequency with which bronchitis supervenes on Pulmonary Vesicular Emphysema, it must be remembered that the blood of the bronchial arteries is returned to the heart chiefly through the pulmonary veins, and that many good observers affirm that the bronchial mucous membrane is in great measure nourished by the blood of the pulmonary artery, and that anastomoses exist between the finest divisions of the bronchial and pulmonary arteries.

The next most frequent determining and permanence-securing causes of Large-lunged Vesicular Emphysema are diminished ordinary or habitual expiratory force, dependent on age-degeneration of the bones and cartilages in the thoracic parietes, without loss of full muscular inspiratory power, occurring alone, or more commonly conjoined with thickening of the tissues and diminished elasticity of the lungs—changes also due to age-degeneration.

As bronchitis, winter cough, and congestion of the lungs are common at advanced periods of life, *i.e.*, at the period of life when, without loss of muscular inspiratory power, age-degeneration of the bones and cartilages of the thorax and of the lungs is common, it is manifest that violent expiratory efforts with closed glottis, habitually defective expiration, and therefore excessive inspiratory dilatation, changes in the lung due to congestion, and changes in the lung-tissue due to age-degeneration, must in a very large number of cases conspire to produce Large-lunged Vesicular Emphysema.

FIG. 1 shows increase in the thickness of the walls of the air-cells. Magnified two diameters.

The changes which occur in the texture of the lung, in consequence of continued congestion, have been admirably described and figured by Rokitansky.

FIG. 2.—A portion of the above magnified 400 diameters.

When the lung is congested, as from disease of the left side of the heart, an increase in the quantity of the connective tissue occurs, the

walls of the air-vesicles are thickened, the parenchyma appears thicker and swollen and unusually resistant.

On section of the lung, the margins of the lung-vesicles are thicker than in health, and the cavity of each vesicle more visible than it should be, because its thickened walls prevent collapse. Sometimes the cavity of each vesicle is increased, and the lungs are larger than they should be; in other words, the substance of the lung is toughened and thickened from the formation of tissue, and enlargement of the lung, with dilatation of the vesicles, follows when any of the determining causes of over-distension of the air-vesicles come into action.

But, however produced, permanent over-distension of the air-vesicles is followed by various pathological changes in their walls. Some of these changes are the direct mechanical result of their over-distension; some are the result of degenerative changes in the structures thus mechanically injured; some of defective nutrition consequent on the injury inflicted on the capillaries of the walls by their stretching; some of altered nutrition due to the alterations in structure; some are due to the pathological states to which the permanence of the over-distension is owing. So that when the disease is far advanced, and has existed for some time, not only are individual air-cells enlarged, but the partitions between many are perforated; between others they are reduced to mere ridges; at places they have altogether disappeared; and at places they are greatly, though it may be irregularly, thickened by imperfectly constituted fibrous tissue formed in and about the normal structures. And so, ultimately, atrophy of some structures is conjoined with increase in size and thickness of others; and rarefaction and condensation may affect adjacent parts of the same lung.

If a portion of lung in an advanced stage of Vesicular Emphysema be in-

Fig. 3.—Section of lung in an advanced state of Chronic Vesicular Emphysema. (From Rokitansky, Lehrbuch der Pathol. Anatomie. B. iii. 1861.)

flated, dried, and then cut across, the cut surface appears to be made up of spaces varying in size from a millet-seed to a hemp-seed, while near to the apex and margin of the lungs may be a few much larger spaces. These spaces or small cavities are separated and intersected by septa and by threads of very variable degrees of thickness.

Black pigment accumulates in considerable quantity on the inner surface of the dilated vesicles, and amid the fibrous and other solids,

This black pigment owes its origin in part to the conversion of the hæmatin in the partially destroyed capillaries into melanin.

When the whole of both lungs are emphysematous, the changes just described are much more advanced at the margins and apices than they are elsewhere; and, as a rule, they are more advanced at the base of the left, than they are at the base of the right lung; these being, in the great majority of cases, the parts first to suffer in Large-lunged Vesicular Emphysema, because they are the parts least compressed and least supported during expiratory efforts with closed glottis.

Effect of over-distension of the air-vesicles on the circulation.—The capillaries of the pulmonary artery distributed on the walls of the air-vesicles are at first stretched in proportion to the over-distension of the vesicles, and then, the over-distension continuing, some of the stretched vessels give way and are obliterated.

The passage of blood through the capillaries lengthened by stretching, must be attended by increased friction, in proportion to the lengthening and narrowing of the vessels.

Destruction of the capillaries diminishes the number of channels through which the blood can pass, and so impedes, in proportion to the number of capillaries torn, the passage of the blood from the right to the left side of the heart.

Impediment to the flow of blood through the lungs is the cause of the greater number of the primary and secondary symptoms of Large-lunged Vesicular Emphysema.

The several causes of impediment to the flow of blood through the lung and their modes of action are—

1. Deficient extent of chest-movement in ordinary respiration; especially deficient ordinary or habitual expiratory movement:

2. Violent expiratory efforts with closed glottis; by the pressure brought to bear on the heart and great vessels, as well as on the air in the interior of the air-vesicles, and so on the capillaries in their walls:

3. Diminished resistance from loss of elasticity of the lung; by disturbing the normal proportion borne by the pressure of the air on the inner to that on the outer chest-walls:

4. Lengthening of the pulmonary capillaries; by increasing the friction:

5. Destruction of pulmonary capillaries; by diminishing the channels for the passage of the blood from the pulmonary artery to the pulmonary vein.

As the establishment of an efficient collateral pulmonary circulation is anatomically impossible, any impediment to the flow of blood through all, or nearly all, the pulmonary capillaries, must have as direct result impediment to the escape of blood from the right ventricle.

The first effect of difficulty to the passage of blood through the pulmonary capillaries must be, in accordance with general laws, increased efforts, and so hypertrophy of the walls of the right ventricle; increased pressure on the inside of the right ventricle, and so dilatation of its cavity.

At the outset, the impediment to the onward passage of the blood may at parts of the lung be trifling, compared with the impediment at other parts; in such case these parts suffer from the increased blood-pressure, become hyperæmic, and, it may be, œdematous.

The impediment to its onward passage is soon felt by the blood in the right auricle, and in the whole systemic venous system, of which the right heart is merely the terminus. When the auricle and ventricle are dilated, the right auriculo-ventricular orifice is dilated, and the result of increase in its circumference, without corresponding increase in the size of the tricuspid valve, is incompetence of the valve to close the dilated opening, and regurgitation of blood during the ventricular systole, from the right ventricle to the right auricle, and veins opening into it.

But the impediment to the flow of blood through the pulmonary capillaries is not only followed by over-distension of the venous system, but ultimately the blood passes from the systemic capillaries into the veins with difficulty, and so an impediment arises to the escape of blood from the arteries, and from the left side of the heart, which is merely the head of the general arterial system.

That such impediment to the escape of blood from the arteries does exist when there is strong impediment to the flow of blood through the lungs, is manifested by placing the finger on an artery when a patient suffering from General Pulmonary Vesicular Emphysema coughs violently; the artery instantly becomes full and tense, and, for the second of violent expiratory effort, ceases to pulsate.

Over-filling of the capillaries of an organ or tissue with retardation of the flow of blood through them never continues for any length of time, and is never repeated frequently without inducing changes in the structure of that congested organ or tissue.

The changes of the several organs, resulting from mechanically-induced congestion, are considered at length in the articles on diseases of special organs. Only such changes of special organs as give rise to the more important symptoms in bad cases of Large-lunged Pulmonary Vesicular Emphysema, will be here considered.

Speaking generally, if an organ be the seat of mechanically-induced intermitting congestion, the earliest result is increased nutrition and enlargement of the organ. When the dilatation of the capillaries has reached a certain degree and becomes permanent, then wasting of the structures of the part with increase in connective tissue, especially of imperfectly-formed connective tissue, may result. The formation of the latter may precede, and greatly preponderate over the wasting of the natural structures of the part.

A large number of free granules, of olein and protein, are found among the proper anatomical elements of the part almost from the very commencement of the congestion; and fatty degeneration of the normal structures frequently precedes their disappearance.

The parts that especially suffer in Large-lunged Vesicular Emphysema are—

The Heart.—First and most certainly, the heart.

The first effect of the impediment to the passage of the blood through the lungs is increase in the muscular tissue of the right side of the heart; then follow accumulation of blood in the ventricle, and some dilatation of its cavity. The right auricle next suffers in the same way, and soon the whole venous system: the veins of the heart suffering over-distension in common with the other veins.

Mechanically-induced congestion of the walls of the heart, with increased action of the organ, leads not only to hypertrophy, but ultimately to induration and toughening of the walls. When these changes have occurred in its muscular tissue, the heart loses its power of close contraction, and permanence of the dilatation produced by the pressure of the blood on its inner surface, is the result.

Free granules of olein and protein are found between the muscular fibres; and, after a longer or shorter time, fatty degeneration of the damaged muscular tissue follows.

When distension of the veins has reached a certain point, the blood escapes from the systemic capillaries with difficulty, and increased action of the left ventricle follows. As the walls of the left side of the heart suffer from the same mechanically-induced congestion as the walls of the right side, when impediment to the escape of blood from the left ventricle is established, its walls and cavity experience, though in a less degree, the same changes in texture, &c., as the right side of the organ, viz., hypertrophy, induration, toughening, and permanent dilatation.

The Liver.—The radicles of the hepatic vein, then the terminal twigs of the portal vein, and finally its radicles suffer congestion from the same cause as the systemic capillaries, *i.e.*, from the impediment to the escape of blood from the inferior vena cava.

In consequence of the impediment to the circulation, the liver is first enlarged from mere congestion, and in this stage a variety of " nutmeg liver" is found after death.

When the congestion has continued for some time, the organ is more or less enlarged, indurated, and toughened, and free granules of olein and protein infiltrate all its tissues; then its natural structures waste, especially, it is said, the cell element, and a certain amount of granular atrophy is the final result.

Ascites very rarely occurs before the hepatic structure is organically injured, and rarely to any great amount from those changes only which follow directly from the impediment to the circulation here considered.

The Kidneys, in common with other organs, suffer congestion in cases of extreme Large-lunged Vesicular Emphysema.

This extreme congestion is evidenced during life by the presence of albumen, and sometimes of blood, in the urine.

The kidney suffering from mechanically-induced congestion is at first larger, darker, and moister than in health. Granules of olein and protein are scattered through all its structures. After a time,

induration and toughening of the organ follow. A slight amount of granular atrophy of the previously enlarged kidney is the ultimate result.

The Connective or Cellular Tissue throughout the body suffers from its mechanically-induced congestion. Its texture is toughened and thickened, and serosity is effused into its meshes.

Anasarca is one of the earliest consequences of over-filling of the venous system from impediment to the flow of blood through the lungs. The anasarca is frequently attributed to the regurgitation of blood through the right auriculo-ventricular orifice; but both the regurgitation and the anasarca are really due to a common cause, *i.e.*, to the state of the pulmonary capillaries. As a rule, however, before over-distension of the veins is so great as to relieve itself by letting out serosity into the cellular tissue, the pressure on the inside of the right ventricle and auricle is sufficient to dilate the auriculo-ventricular orifice to such an extent, that the tricuspid valve is incompetent to its closure, pulsation in the jugulars is perceptible, and the anasarca is then erroneously attributed to tricuspid regurgitation, as it is often called.

Blood and General Nutrition.—Niemeyer has pointed out that congestion of the venous system from mechanical impediment to the onward flow of blood through the lungs, or right heart, cannot exist without causing impediment to the escape of its contents from the thoracic duct. To this Niemeyer attributes the deficiency of fibrine and of albumen in the blood in cyanosis dependent on mechanically-induced over-filling of the venous system, and to it he also attributes the general emaciation which occurs in advanced cases of Pulmonary Vesicular Emphysema.

The Vessels of the Lungs.—In the last stages of the disease, after the left ventricle has suffered hypertrophy and dilatation, secondary lesions of the lung not unfrequently occur—thus the lungs may become greatly congested, and œdema of the lungs or congestive pneumonia follow. The mechanical impediment to the flow of blood through the pulmonary capillaries has told back through the systemic capillaries on the left side of the heart, and so on the radicles of the pulmonary veins.

Symptoms of Large-lunged Vesicular Emphysema.—The chief direct symptoms of Large-lunged Vesicular Emphysema are—

(*a*) Increase in the size of the thorax;

(*b*) Increase in the resonance of the thorax, and prolonged expiration;

(*c*) Shortness of breath.

(*a*) The lungs are larger than in health, and the capacity of the thorax is in proportion to the size of the lungs.

The increase in the circumference of the thorax is effected chiefly by diminution in the natural obliquity of the ribs.

By this alteration in the direction of the ribs, the lower intercostal paces are very considerably widened.

The sternum is carried **forward.**

The lower latero-dorsal bulging of the thorax is increased.

The enlargement of the circumference of the chest thus gained is made still greater by posterior curvature of the lowest cervical, the dorsal, and upper lumbar part of the spinal column. The patient stoops, he grows round-shouldered and round-backed.[1]

Increase in the capacity of the thorax from above downward is produced by lowering of the diaphragm. At the termination of expiration in extreme cases of Large-lunged Vesicular Emphysema the diaphragm lies very low, so that it is not in contact with the inner surface of even the lowest rib.

When the air-vesicles of the upper half of the lungs are the first to suffer over-distension, or are much more affected than are the air-vesicles of the lower part of the lung, the upper part of the thorax is disproportionately larger. When the determining cause of the over-distension has been violent cough from bronchitis, then the disproportion in size between the upper and lower part of the thorax is sometimes increased by imperfect expansion of the lower part of the lungs ; the condition of the bronchial mucous membrane and the contents of the bronchial tubes preventing the free and ready entrance of the air into the air-cells of these parts of the lung.

The increase in the capacity of the thorax is determined by the forces which determine the over-distension of the air-vesicles, viz., by repetition of full inspiratory efforts, expiratory efforts with closed glottis, and diminished elasticity of the thoracic parietes, or of the lungs, or more commonly of the two conjoined.

(*b*) Of the physical signs, after those furnished by inspection of the thorax, by far the most constant and important in regard of diagnosis, is increased resonance on percussion—clear full sound. The abnormal clearness on percussion is due to the relative increase in the quantity of air in the chest, and to the tension of the chest-walls.

As the large lungs overlap the heart, the region of precordial dulness is diminished, and as the diaphragm is flattened, the hyper-resonance extends posteriorly even to the twelfth rib, and in front often as low as the margin of the thorax, even on the right side, the liver lying altogether under the abdominal parietes.

Expiration is, in extreme cases, considerably prolonged in consequence of the diminution in the resilience of the chest-walls and lungs, and of the large size of the latter. At the same time, the inspiratory murmur is short and feeble. But when this form of Pulmonary Vesicular Emphysema is limited to a part of the lung, the only physical signs are local bulging and hyper-resonance.

(*c*) Shortness of breath is always present in Large-lunged Vesicular Emphysema. At first, the shortness of breath is only felt on exertion ;

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[1] Whenever the depth of the chest, from before backwards, requires to be increased, *e. g.*, in dilatation of the heart—effusion into the pericardium—the patient instinctively rounds his back and elevates his shoulders.

the patient cannot mount a hill as he did. Then, when walking on level ground, he requires to stop, from time to time, to take in breath,—he breathes too frequently, and pants a little ; or it may be that he " suffers with his breath " after a full or an indigestible meal, when the descent of the diaphragm is impeded by a distended stomach. However the shortness of breath is induced, the subject of Large-lunged Vesicular Emphysema is, from a very early period, conscious that his " wind " is no longer what it was.

As the disease advances, the shortness of breath is experienced on the least exertion, *e.g.,* ascending a few steps, or a gentle slope ; and finally, even when sitting on a chair. The patient is always panting.

By the altered position of the ribs and the diaphragm, a considerable increase in the capacity of the thorax is, as has been shown, obtained, but it is obtained at the expense of the inspiratory capability. The chest-walls are constantly expanded, and when the disease is far advanced, the capacity of the chest may be greater at the termination of expiration, than, in the normal condition of the lungs and chest-walls, it should be at the termination of inspiration. As Dr. C. J. B. Williams has tersely expressed it, " Breath is taken as it were on the top of breath."

The lowering of the diaphragm may be so considerable, it is said, as to cause its physiological action to be reversed. In place of increasing the capacity of the thorax by its contraction, the diaphragm may draw, it has been said, the lower ribs inwards, and so diminish to a slight extent the capacity of the lower part of the chest at the end of inspiration.

The diaphragm may be forced downwards by the expiratory efforts, which determine over-distension of the air-cells, but it probably never lies very low till the elasticity of the lungs is considerably impaired.

The great natural agent in effecting the ascent of the diaphragm, after it has been lowered by its own contraction, is the elasticity of the lungs. The muscles relax at the termination of inspiration, and the diminution in the size of the lungs resulting from their resilience, greatly aids in determining the passive ascent of the diaphragm. When the lungs, from loss of elasticity, no longer diminish in size as much as they should, at the termination of inspiration, the ascent of the diaphragm is less than it should be, and it begins to act at the commencement of each inspiration from a lower and lower level; consequently, the increase in the capacity of the thorax obtainable by its contraction is always lessening, till finally, it is perhaps just possible that its normal physiological action may be, as above stated, reversed.[1] When the ordinary muscles of inspiration are, in consequence of the permanent expansion of the chest, unable to

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[1] The common cause of recession of the lower part of the chest during inspiration is some impediment to the free entrance of the air into the lungs, and the pressure of the external air for this reason being brought to bear with undue force on the outside of the thorax by the powerful action of the inspiratory muscles. The lower parts of the chest-

dilate it sufficiently to take in a proper supply of air, all the extra-ordinary muscles of inspiration are habitually employed in breathing; hence the muscles of the neck, back, &c., capable of aiding inspiration are, after a time, considerably hypertrophied, the shoulders are raised, and the enlargements of the muscles of the neck, the scaleni especially, give a peculiar breadth to the neck.

Imperfect aëration of the blood resulting from the damaged state of the pulmonary capillaries, and the changes which take place in the walls of the air-vesicles after they have been long over-distended, add greatly to the shortness of breath; while the dilution of the air taken in at each inspiration by the large quantity of residual air left after expiration, must still farther distress the breathing by interfering with aëration of the blood.

The shortness of breath, then, in uncomplicated Large-lunged Vesicular Emphysema, is due to the small extent of movement of the chest-walls, including the diaphragm, during respiration, to the im-purity of the air in the thorax at the termination of inspiration,[1] to the state of the capillaries of the pulmonary artery, and to the structural changes in the substance of the walls of the air-vesicles.

If bronchitis in any form, or asthma supervene, the distress of breathing is greatly increased; and in some cases in which the dis-tress of breathing has been unusually great, fatty degeneration of the heart has been found after death (Virchow).

General description of the symptoms in a case of advanced Large-lunged Vesicular Emphysema. — The thorax is barrel-shaped; the antero-posterior, lateral, and vertical diameters are increased; the sternum is arched; the lower cervical, dorsal, and upper lumbar spine is curved, concavity forward; the ribs are too horizontal; the intercostal spaces are widened, and but little, if at all, depressed below the level of the ribs; the posterior bulgings on either side of the vertebral column are greater than they should be; the costal angle is larger than in health, and as the diaphragm is flattened and the lower part of the sternum is forced forward, at the same time that both lungs are enlarged, the heart is at once less covered than in health by the sternum, thrust downwards by the forces that over-distended the air-vesicles, and carried downwards by the contraction of the diaphragm, and can, in consequence, be felt and seen beating

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walls are there most yielding, and are therefore pressed inwards by the weight of the atmosphere. On this and other points connected with the deformity of the chest in Pulmonary Vesicular Emphysema, the reader is referred to Dr. Sibson's elaborate and most able paper in the thirty-first volume (1848) of the Medico-Chir. Soc. Trans., "On the Movements of Respiration in Disease."

[1] Although the capacity of the chest is greater in Large-lunged Vesicular Emphysema than it is in health, spirometrical observations show that its vital capacity, as measured by the quantity of air that can be expelled after deep inspiration, is diminished. The residual air must therefore be much greater than it should be. Speaking of the difficulty of breathing in Emphysema, Magendie (Leçons, 1825, tome i. p. 169) observes:—"The tissue of the lung has lost some of its elasticity, and no longer reacts with sufficient force on the air which has penetrated into its parenchyma."

below the ensiform cartilage. The heart, and especially the right ventricle, is dilated, and hypertrophied — its impulse is heaving, and its dilatation and hypertrophy render the epigastric pulsation very perceptible.

The shoulders are raised, and the muscles of the neck and shoulders, especially the sterno-cleido-mastoidei, the scaleni, the omo-hyoid, and the trapezii, stand prominently out.

The fossa behind the clavicle is frequently deepened ; when, however, there is excess of Emphysema above the level of the first rib, there may be post-clavicular bulging. Under all circumstances, when the patient coughs there is undue prominence, or bulging even of the post-clavicular fossa, and of the intercostal spaces, the air being forced from the more to the less compressed and supported parts by the expiratory efforts preceding the opening of the glottis.

The neck is broad from hypertrophy of its muscles, and its veins are unduly prominent. As the obstruction to the circulation increases, the veins of the neck pulsate synchronously with the beat of the right ventricle, and fill from below when emptied by the pressure of the finger. The whole venous system is manifestly dilated, the larger veins of the upper extremities have a knotted appearance from over-distension just above their valves, the hæmorrhoidal veins are enlarged, thickened, &c., and often bleed— it may be to the great relief of many of the discomforts from which the patient is suffering. The face has a coarse, bloated, dusky, and, on exertion, even livid aspect ; the alæ of the nose, and the lips, especially the lower lip, are thickened. The eyes are prominent, the conjunctivæ injected, occasionally yellowish, and the eyelids puffy—drowsiness, mental dulness, and headache are common symptoms. Emaciation is sometimes very considerable. The legs are œdematous, or the whole cellular tissue the seat of anasarca. Orthopnœa is often present, because in the recumbent position the extraordinary muscles of inspiration can have only imperfectly supported points, in place of fixed points, from which to act ; and again, because the weight of the body in the recumbent position interferes with the expansion of some part of the chest-walls, and the position and weight of the abdominal viscera with the descent of the diaphragm.

In this stage of the disease the urine frequently contains albumen, and now and then blood and blood-casts of tubes.

The abdomen generally is fuller than natural. The spleen and liver are increased in size, and the latter organ is frequently so much depressed by the determining cause of the distended lungs, by the enlarged and distended heart, and by the flattened diaphragm, that its upper convex surface can be distinguished by eye and touch through abdominal walls.

When from supervention of bronchitis, or other cause, the impediment to the pulmonary capillary circulation is temporarily increased, the liver and spleen may be proved, by percussion and touch, to be

larger than before, and to resume their former size, as the circulation through the lungs becomes freer, and the mechanically-induced congestion is in consequence lessened.

The pulse in Large-lunged Emphysema is often small and weak, from the small quantities of blood which pass through the lungs and therefore into the left ventricle.

The urine is, speaking generally, that of imperfect respiration, and of congestion of the kidneys and liver. At times it is very abundant, pale, clear, and of low specific gravity; at others it is scanty, high-coloured, and loaded with lithates, which as the urine cools form a heavy brickdust-like sediment. This deposit is not in all cases due merely to the concentration of the urine, there may be an absolute increase in the quantity of uric acid. Owing to the imperfect aëration of the blood there is a scanty supply of oxygen distributed through the system, hence the products of tissue metamorphosis are in a lower stage of oxidation, and uric acid is formed to some extent in place of urea. Parkes thinks it is only when bronchitis is superadded to Emphysema that there is such deficient oxidation as to lead to excess of uric acid in place of urea. J. C. Lehmann, in a carefully-observed case, found the urine after each attack of difficulty of breath deficient in urea and very acid. It contained oxalic acid and allantoin. To this Parkes objects, that in Lehmann's case bronchitis complicated the Emphysema, and refers to a case of uncomplicated Emphysema so severe as to cause cyanosis and constant dyspnœa, observed by Ranke and himself, in which very little uric acid and a full quantity of urea were present in the urine. Biemer says, after quoting the observations of Lehmann and Ranke, that he has more often been able to detect small quantities of bile pigment in the urine.

A trace of albumen may, when the disease is far advanced, be constantly present in the urine; the quantity being increased with every increase of the impediment to the flow of blood through the lungs. When the congestion of the kidneys is suddenly greatly increased, or attains, even slowly, an extreme degree, the urine contains blood and blood-casts of tubes. Much albumen, with little evidence of impediment to the flow of blood through the lungs, renders it probable that organic disease of the kidney is present.

It is not uncommon for the symptoms to be very trifling for some years, and then for a year or more, to see the graver symptoms only when the patient has an attack of bronchitis; with the cessation of the bronchitis the œdema of the legs, the albumen in the urine, and the jugular pulsation frequently disappear. The over-distended heart and veins having their walls, as yet, to any serious degree, undamaged, contract nearly to their normal dimensions, when the extra impediment to the flow of blood through the lungs, due to the acute attack, has passed away. But the improvement is only for a time, another attack of bronchitis renews the serious symptoms, and after one or more such renewals, they are permanently established.

The variations in severity of the chief symptoms of Large-lunged Vesicular Emphysema may be summed up thus: the increase in size of the thorax varies from that obtained by a slight diminution in the natural obliquity of the ribs, or trifling local bulging, to the utmost expansion of the chest-walls; the hyper-resonance on percussion, from slightly increased clearness to the fullest clear sound; the prolongation of expiration, from an amount difficult to appreciate, to that in which it considerably exceeds in length the inspiratory sound. The impediment to the flow of blood through the lungs varies, from just enough to give, when the patient coughs, undue prominence to the great veins of the neck, to sufficient to cause hypertrophy and dilatation of the right side of the heart, jugular pulsation, and knotting and enlargement of all the superficial veins, anasarca, albuminuria, enlargement of the liver and spleen, dilatation of the systemic capillaries and arteries, hypertrophy and dilatation of the left side of the heart, and finally organic changes in the structure of all the organs in the body, and of the connective tissue generally. The shortness of breath varies from a mere "touch in the wind" to inability to move without great distress of breathing.

The imperfect aëration of the blood varies from just sufficient to cause a slightly dusky hue of the lips on exertion, to enough to give the patient the purple or leaden hue of cyanosis.

When large-lunged Vesicular Emphysema is limited to a lobe or part of a lobe, as not unfrequently happens, then local fulness or bulging, and hyper-resonance with trifling shortness of breath, are commonly the only evidences of the disease.

SMALL-LUNGED (OR ATROPHOUS PULMONARY) VESICULAR EMPHYSEMA.—After Large-lunged Vesicular Emphysema has lasted some time, and the over-distension of the vesicles is extreme, a certain, it may be considerable, amount of wasting of the tissues of the lung ensues; and thus a form of Atrophous Emphysema is established.

But in the variety of Emphysema designated Small-lunged or Atrophous Pulmonary Vesicular Emphysema, atrophy of the lung-tissue is the primary disease, or it supervenes on trifling primary over-distension.

Small-lunged Vesicular Emphysema is confined to persons well past middle-life. Those who suffer from it are commonly thin. Withered-looking, shrivelled, old persons frequently have their lungs damaged by this form of Emphysema. It is a far less troublesome and less grave affection than is Large-lunged Vesicular Emphysema.

In primary general Small-lunged Vesicular Emphysema the whole of both lungs suffer. There is waste of tissue, true atrophy. In some cases fatty degeneration has been said to precede absorption, or final disappearance of tissue.

In this form of Emphysema the separate vesicles are not dilated; but the partitions between adjacent vesicles with their pulmonary

capillaries and other structures disappear or are reduced to mere threads, and two or more vesicles are thus thrown into one. No over-distending force is necessary to determine the increased capacity of the vesicles.

Lungs the subject of this disease are smaller, lighter, and drier than are healthy lungs. They would, from the destruction of the capillaries, be pale, but the pallor from this cause is commonly concealed by the large amount of black pigment spread through them. They weigh much less than healthy lungs, because they have lost much of their natural structures. The air-vesicles are large, but the lungs themselves are small.

The division between the superior and inferior lobe is more vertical than in health. The elasticity of the lungs is in a great measure lost—there is no resilience in them—they pit on pressure, and the pit remains. Their small size, their lightness, and the *very* small space into which they may be compressed are often most remarkable. They are occasionally so much wasted, that, on opening the thorax, they sink back at once toward the spine and posterior part of the thorax. When the lungs and air-passages are in health, death takes place at the termination of expiration. On opening the thorax, healthy at the moment of death, and permitting the pressure of the air to bear on the outer surface of the lungs, there is at once a slight diminution in their size. This diminution is due to the elasticity of the lungs. Before the opening of the thorax the complete elastic contraction of the lungs was opposed by the pressure of the air on the inner surface of the air-vesicles.

In Hypertrophous Pulmonary Vesicular Emphysema the resilience of the lungs is diminished, hence when the thorax is opened there is less contraction of the lungs, and therefore less separation of the lungs from the chest-walls, than there is when the lungs and air-passages are healthy. The quantity of solid tissue constituting the walls of the air-vesicles, &c., and the irregular thickening of that tissue, prevent any mere collapse of the lungs.

When the lungs are in a state of extreme Atrophous Vesicular Emphysema, they have not only lost in a great measure their elasticity, but a large quantity of their solid tissues has disappeared. The consequence is that, when the thorax is opened, and the pressure of the air on the internal and external surfaces of the lung is equalized, although little or no diminution may occur from its resilience, the weight of the lung may be sufficient to cause it to fall in like an inflated bag of wet paper.

If the subject of extreme Atrophous Pulmonary Vesicular Emphysema suffer from cough, then Local Emphysema with large vesicles is frequently superadded to the General Atrophous Emphysema. The elasticity of the lungs being diminished, the vesicles of the parts least compressed and least supported during expiratory efforts being permanently and greatly over-distended, atrophy of their walls throws several air-vesicles into one, and air being forced into the large

cells so formed may lead to their extreme dilatation. For reasons previously assigned, these vesicles are found at the margins and apex of the lung.

Coincidently with the occurrence of the changes in the textures of the lungs which constitute Atrophous Vesicular Emphysema, the ribs and their cartilages experience degenerative changes by which their elasticity is diminished, but at the same time also the inspiratory muscles shrink and lose strength.

The chief direct symptoms of Small-lunged or Atrophous Pulmonary Vesicular Emphysema are—(1) shortness of breath ; (2) diminution in the size of the thorax.

Shortness of breath in Small-lunged Emphysema is never felt to any notable degree, unless the patient makes exertion ; and as the disease usually occurs in the aged, or in those wasted from other chronic diseases, persons indisposed and incapable of moving quickly, exertion sufficient to cause distressing shortness of breath is rarely made.

Primary Atrophous Vesicular Emphysema is commonly attended by general waste, and is therefore accompanied by waste of blood as well as of tissues ; so that the capillary pulmonary vessels, although reduced in number, still suffice for the passage of the diminished quantity of blood.

Again, the lungs being small, the expiratory power is enough to drive out the air, and the play for inspiration is considerable. The patient, in place of, as in Large-lunged Vesicular Emphysema, always "taking in breath on the top of breath," in Small-lunged Vesicular Emphysema inspires from the bottom of his breath.

The chest in Small-lunged Vesicular Emphysema is diminished in capacity, and all its diameters are less than in health. The diminution in the antero-posterior and lateral diameters is obtained by a great increase in the obliquity of the ribs. The upper intercostal spaces next the sternum are widened and depressed below the level of the ribs. So obliquely placed are the lowest ribs, that their cartilages almost reach the crest of the ilium, and the intercostal spaces are lost, the ribs themselves really touching. The cartilages between the ribs and sternum, as the ribs become abnormally oblique, bend so as to form an obtuse angle. Respiration is short, the thorax moves as a whole in inspiration, and the expiratory recoil follows quickly. The inspiratory murmur is short and feeble—the expiratory not prolonged. From the loss of elasticity in the ribs and cartilages, and the imperfect tension of the chest-walls, the resonance on percussion may be even less than in health, although the solids in the chest are diminished. As the lungs are small, the heart is less covered than it should be, and so the extent of precordial dulness may be increased, and that, although the heart itself may be partaking of the general atrophy. As Atrophous Emphysema is usually accompanied by waste of blood, and as the general muscular power of the patient forbids active exercise, there is commonly in Small-lunged Emphysema not only little distress

in breathing, but no damming back of the blood in the right ventricle, over-distension of the venous system, dropsy, or hypertrophy, or dilatation of the heart.

Complications of Pulmonary Vesicular Emphysema.—The frequent conjunction of bronchitis and Pulmonary Vesicular Emphysema has been admitted from the time the latter was recognised as a special disease. Laennec considered the Emphysema to be in all cases the consequence of bronchitis, and especially of that form of bronchitis which was designated dry catarrh. And Louis, while denying the relation of the two diseases as cause and effect, admitted the frequency of their co-existence.

It is now established that bronchitis is the most common cause of the direct production of Pulmonary Vesicular Emphysema, and also that Emphysema may be developed independently of bronchitis; and that when so established, the subjects of Emphysema are very prone to suffer from bronchitis. So that although, as a rule, bronchitis is the cause of Pulmonary Vesicular Emphysema, it may be the consequence; and the Emphysema, in rare cases, may be unaccompanied from first to last by bronchitis.

One cause of the frequency with which bronchitis supervenes on Pulmonary Vesicular Emphysema is, that when a part of the lung only is emphysematous, and the passage of the blood through the capillaries of that part is consequently impeded, hyperæmia of the non-emphysematous tissues is the necessary result—the blood passing into the vessels of the part which offer it the less resistance.

Chronic general catarrhal bronchitis, with much violent cough and little expectoration, is the most common complication of general Large-lunged Vesicular Emphysema. Acute capillary bronchitis is one of the most common causes of death in the same form of the disease. If the heart be, as it so often is, dilated and hypertrophied, then the acute capillary bronchitis is usually attended with much congestion of the substance of the lung.

Chronic bronchitis with profuse purulent expectoration is less common in Large-lunged than it is in Atrophous Pulmonary Vesicular Emphysema. When the purulent discharge is considerable, the so-called amyloid degeneration of various organs is said to occur pretty frequently. In this case, amyloid degeneration is connected with the profuse suppuration and not with the Pulmonary Vesicular Emphysema.

Dilatation of the bronchial tubes is common in all forms of Pulmonary Vesicular Emphysema.

In severe Large-lunged Vesicular Emphysema, it is common to find an excess of fluid in the pericardium after death. It is only when congestion and œdema of the lungs have complicated the disease that an excess of serosity is found in the pleuræ.

The subjects of Large-lunged Vesicular Emphysema frequently suffer from disturbance of the digestive organs. The liver is loaded with blood, and the bile formed is thick and dark. The walls of the

stomach are congested, and ultimately thickened. The result is notable disturbance of the stomach digestive processes. The patient suffers from visible distension of the epigastric region, and also from a sense of weight and fulness in the same part, especially after food, altogether disproportionate to the quantity and the quality of the food taken. Flatulence and acidity of stomach are troublesome symptoms.

The distension of the stomach is frequently so great as very decidedly to intensify, by the impediment it offers to the descent of the diaphragm, the habitual dyspnœa. The derangement of the stomach is also a not infrequent exciting cause of asthma.

Spasmodic asthma is a very common complication of Pulmonary Vesicular Emphysema, especially of the large-lunged form of the disease. Attacks of extreme difficulty of inspiration supervene suddenly in the early morning, or at uncertain times, on the habitual shortness of breath. In addition to attacks of ordinary spasmodic asthma, the subjects of Pulmonary Vesicular Emphysema often suffer for weeks together from increased dyspnœa, out of all proportion to any catarrhal bronchitis which may happen to be present. This dyspnœa is, in a great measure at least, due to spasm of the bronchial muscular fibres.[1]

Phthisis.—One of the most marked anatomical characters of congenital tuberculosis, *i.e.* of an inherited disposition to the deposit of tubercle, is the small size of the lungs. It is by no means uncommon for a deposit of tubercle to take place in the apex of such lungs, and then for the tubercle to obsolesce or to calcify. The subjects of these changes frequently live to an advanced age. Chronic solidification with contraction of the apex of the lung, much black pigment in the solid tissue, and Local Emphysema with large vesicles, frequently follow the obsolescence or calcification of the tubercles.

After passing middle life, the subjects of these lesions frequently become affected with general Atrophous Pulmonary Vesicular Emphysema, rarely, if ever, with Large-lunged Vesicular Emphysema. Subsequently, the solid tissue of the apex of the lung may undergo molecular disintegration, and a cavity be formed; death, in such cases, is said to have occurred from tubercular consumption, when, indeed, there are no tubercles present. This is a form of senile phthisis.

Headache and drowsiness are common consequences of large-lunged Vesicular Emphysema. These symptoms are due to congestion, the consequence especially of the mechanical impediment to the passage of blood through the lungs, aided in some cases by the imperfect aëration of the blood, and by those changes in the coats of the vessels so commonly seen in the subjects of the disease here considered. The veins and arteries, from changes in their walls, partly due to the congestion of their *vasa vasorum*, lose some of their normal

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[1] It is possible that in many cases asthma precedes Pulmonary Vesicular Emphysema, the violent efforts to inspire are the determining causes of the Emphysema, that in as in so many other cases the effect has been mistaken for the cause.

reactive force, and hence the pressure of the blood on the inside of their walls leads here, as elsewhere, to their permanent dilatation. After death, we find not only increased capillary vascularity and excess of serosity in the meshes of the pia mater, but the arteries and veins in the same structure manifestly larger than they should be.

Diseases which in their progress are frequently complicated with Pulmonary Vesicular Emphysema.—All diseases accompanied by severe cough, by diminution of the whole or part of a lung, or by impediment to expiration, are accompanied by over-distension of the air-vesicles.

All diseases or changes in nutrition attended by damage to the elasticity of the lung, render permanent what would otherwise be transient over-distension of the air-vesicles.

Winter cough, catarrhal, and other forms of bronchitis, are undoubtedly the diseases most frequently conjoined with Pulmonary Emphysema.

Disease of the left side of the heart, by damming back the blood in the lungs, and so inducing changes in their texture, leads to permanent dilatation of the air-vesicles when their over-distension has been once determined by cough, &c.

Pneumonia is sometimes attended by Acute Vesicular Emphysema of the air-admitting vesicles, but as the walls of the over-distended vesicles are healthy, and their over-distension is neither extreme nor of long duration, they return to their natural size when the pneumonia subsides. After solidification has passed away, the textures of the lung damaged by the pneumonic exudation may be the seat of permanent Chronic Emphysema.

When one pleura contains sufficient fluid to render the lung altogether useless, the opposite side of the thorax expands more than it should, and is for the time larger than in health, and its air-vesicles are enlarged in proportion to the degree of the expansion of the side. Should the impervious lung be, from long compression or other cause, so damaged as never again to admit any quantity of air into its vesicles, then permanent over-distension of the vesicles of the opposite lung is the consequence. It rarely happens that the distension of the air-vesicles in this case is sufficient to interfere with the capillary circulation on their walls, or to induce atrophy or other serious changes of the vesicular septa.

In chronic phthisis, the bases of the lungs very frequently suffer from vesicular emphysema; this is especially likely to happen when there has previously been dry pleurisy with adhesions at the same point. Hyper-resonance of the bases of the lung is, therefore, frequently conjoined with tubercular dulness of the apices. The expiratory efforts of cough are the determining cause; the damage inflicted on the textures by the pleurisy is a common cause of the permanence of the over-distension. The Vesicular Emphysema in this and similar cases, when tolerably limited in extent, is not attended with notable disturbance of respiration or circulation. It must, it is true, add

a little to the shortness of breath, and a little to the impediment to the circulation through the lungs, but these additions are insignificant in comparison with the primary disturbances of respiration and circulation resulting from the original disease. Diseases attended by incomplete occlusion of the air-passages frequently have, as consequence, over-distension of the air-vesicles of the whole or of part of the lung.

In accidental occlusion of the larynx, Acute General Vesicular Emphysema is frequent. Thus, in the case of a woman, who in a state of drunkenness choked herself by thrusting the food with her finger from the over-full pharynx into the larynx, the lungs were the seat of extreme General Acute Emphysema.

In such cases, supposing the obstruction to be at first incomplete, the sufferer does what he would do if the obstacle were removable —he makes the deepest possible inspiration, and then coughs. The violent expiratory effort drives the air into the less compressed and supported parts. The local obstacle to the escape of air being irremovable, causes an excess of air to be retained in the lungs. A second deep inspiration follows, and so finally general over-distension of the air-vesicles is established.

The full normal distension of the lungs with air may be mistaken for Acute General Emphysema, if death occur while the lungs are distended by a deep inspiration, and a foreign body in the larynx prevent the expulsion of the air from the lungs by the natural death-expiratory act.

In croup, the false membrane in the larynx may, in rare cases, act for a short time as a valve, admitting the air to pass into the lung, but opposing its escape, and so cause Acute Vesicular Emphysema.

In croup, again, pulmonary lobular collapse and lobular pneumonia are both common, and, when present, some of the Acute Vesicular Emphysema found after death may be secondary to those lesions of structure.

But more commonly than in either of the modes just enumerated, the Acute Vesicular Emphysema found after death in croup is produced during the expiratory efforts of coughing; that it is so produced is proved by the situations it occupies, viz., those parts of the chest which in these cases are seen during life to recede during inspiration, and to advance during expiration.

In hooping-cough, Vesicular Emphysema is a constant result of the violent expiratory efforts of that disease. When the over-distension is extreme, and is frequently repeated, the elasticity of the walls of the vesicles may be diminished, and then a certain amount of over-distension remains a permanent lesion. As bronchitis is a frequent complication of hooping-cough, disseminated lobular collapse may in some cases aid to a slight extent in the production of the Vesicular Emphysema.

Hereditary nature of Pulmonary Vesicular Emphysema.—Jackson[1] found that 18 of 28 subjects of Pulmonary Emphysema were born

[1] Quoted by Louis.

of parents one of whom was affected with the same disease; while of 50 non-emphysematous patients 3 only came of emphysematous parents; and Niemeyer remarks, " I have known at least one family in which, without catarrh preceding, all the members for three generations suffered from emphysema."

These facts afford some support to the theory that Pulmonary Vesicular Emphysema is hereditary. But Pulmonary Vesicular Emphysema is far too common a disease for Jackson's few oft-quoted observations, or for solitary baldly stated facts, such as that of Niemeyer, to *prove*, or even to render it highly probable that it is really hereditary.

If the cases be excluded in which the disease owes its origin to inherited predisposition to bronchitis, heart disease, asthma, premature age-degeneration, &c., the facts adduced in support of the hereditary nature of Pulmonary Vesicular Emphysema will be reduced to an insignificant figure. It is not denied that the disease may be hereditary, but it is without question in the writer's mind that the evidence adduced in support of its being hereditary in the sense in which tubercle and cancer are hereditary, is altogether insufficient for the proof.

Diseases of the lungs to which Pulmonary Vesicular Emphysema indisposes.—Pneumonia with exudation of lymph, croupose pneumonia as it has been called, rarely occurs in the emphysematous parts of a lung. The destruction of the capillary vessels which occurs in Chronic Vesicular Emphysema, is supposed to afford a certain degree of exemption from this form of inflammation. Although a streak or two of blood in the sputa is not uncommon in the bronchitis from which emphysematous patients suffer, hæmorrhage in quantity from emphysematous lungs is said to be rare. Those suffering from Atrophous Vesicular Emphysema alone enjoy a practical immunity from pneumonia and from hæmorrhage.

Tubercle has been said to be excluded by Pulmonary Vesicular Emphysema.

No doubt active congestion of a part accompanies the formation of tubercle, and active congestion rarely affects the emphysematous parts of a lung, and to the same extent Vesicular Emphysema of the lung indisposes to the deposit of tubercle. It is to the wasting of the vessels in Emphysema of the lung that the immunity, so much as it is, from tubercle is due.

The blood in Pulmonary Vesicular Emphysema is less fully aërated than in health. To this venosity of the blood, Rokitansky attributes the infrequency of tubercle in the subjects of Pulmonary Vesicular Emphysema. His theory, however, is opposed by facts.

TREATMENT OF CHRONIC PULMONARY VESICULAR EMPHYSEMA.— The treatment of Pulmonary Vesicular Emphysema may be divided into Curative, Palliative including the treatment of its direct consequences, and Preventive.

Curative Treatment.—Some therapeutists have supposed that, by the lengthened administration of small doses of strychnine, and others, that by the skilful employment of electrical power, permanent contraction of the walls of the dilated air-vesicles may be obtained. But, although powerful agents for exciting muscular contraction, neither strychnine nor electrical action have any influence in restoring or increasing the elasticity of a tissue. In Chronic Pulmonary Vesicular Emphysema, it is the elasticity of the walls of the air-vesicles which is damaged. Experience, as might have been anticipated, has afforded no evidence in support of the value of the drug or the battery in the cure of Pulmonary Vesicular Emphysema.

Again, some therapeutists have placed persons suffering from Chronic Pulmonary Vesicular Emphysema in a chamber supplied with condensed air, in the hope that the breathing of the condensed air would cure the disease. Others have alleged that great attention to diet, and the administration of iron, and other blood, nervine, and stomach tonics, will, by improving the nutritive powers, cure the disease. But, if it be remembered, that to cure Chronic Pulmonary Vesicular Emphysema of severity sufficient to cause trouble to the patient, is not only to renew the elasticity of the walls of the air-cells, but also to restore the stretched vessels to their normal length and to their natural tone, to repair the apertures in the walls of the air-vesicles, and to replace the torn and otherwise destroyed capillaries by healthy vessels, it will be at once admitted that the cure of Chronic Pulmonary Vesicular Emphysema is impossible. Persons suffering from Emphysema have been greatly relieved by breathing condensed air; but the relief, as might be anticipated, has been temporary only; and while attention to diet, &c., is of no avail to cure the disease, it is of great importance in staying the progress of the disease and relieving distress.

Preventive Treatment.—The great factors of Pulmonary Vesicular Emphysema being—

Excess of pressure of air on the inside of the air-vesicles,

Age-degenerative changes of the parietes of the thorax,

Changes of the texture of the lung, from excess of blood in it,

Age-degenerative changes of the lung,

in order to prevent the disease, and to stay its advance when established, care must be taken to guard against these, its determining and permanence-securing causes. Catarrhal, and all other forms of bronchitis being beyond question the most frequent excitors of the Pulmonary Vesicular Emphysema, the prevention of these diseases is of the very highest importance. To secure immunity from bronchitis, and to prevent its recurrence, clothing must be adapted to the season, and it is necessary that cold and wet, especially fog and cold winds, be avoided. A mild climate has a marked influence in preventing the attack of bronchitis, to which so many are subject during the winter in this country. Hence it is most important for

those whose lungs are the seat of Vesicular Emphysema, to spend the winter in a mild, and not too dry air.

When chronic or sub-acute bronchitis is present, the freer the secretion from the bronchial mucous membrane, and the less violent the cough, the less likely is Chronic Emphysema to follow. Expectorants and opiates combined are the great medicinal agents. The expectorants selected when the cough is dry, should be those that promote secretion ; when the secretion is abundant, those that favour its expulsion.

Violent and irritative cough—that is, cough out of all proportion to the matter to be expectorated, should be restrained by sedatives : Opium, belladonna, stramonium, conium, and prussic acid, are the chief sedative agents in this class of cases. These drugs are more efficacious when given with little water, and in a small quantity of mucilage and syrup. Sedative inhalations are particularly useful. The sedative should be placed on the sponge of Maw's inhaler, and the steam of hot water passed through the sponge. Chloroform vapour exhibited in this way is sometimes very serviceable.

When the secretion from the bronchial mucous membrane is too abundant and purulent, the mineral acids, quinine, iron, especially the tincture of the sesquichloride, and cod-liver oil, are invaluable ; as is also the inhalation of mild stimulants, *e.g.*, iodine diffused in small quantity through the room. In these cases, a change to dry sea-air is often very serviceable.

All efforts which try the muscular powers, as carrying heavy weights, are injurious. All exertions which induce panting, or oblige the person to stop frequently to recover his breath, are calculated to inflict permanent injury. Rapid walking, hill climbing, and violent exertions of all kinds, are to be carefully avoided. Walking exercise should, as much as possible, be limited to level ground.

Many an old gentleman has been hurried to his grave by attempting to follow the birds as he did in his earlier days, and by striving to improve his health by active exercise. It is a great gain for length of life to take old age pleasantly. Those predisposed to the disease and, *à fortiori*, subjects of Pulmonary Vesicular Emphysema, should never attempt to play wind instruments.

When urging these points on a patient, it must never be forgotten that the permanence-securing cause being established, every single over-distension of the air-vesicles permanently increases their size. The increase on each occasion is indeed insignificant ; but as every repetition of the over-distension adds to that previously existing, it follows that, should the over-distension be frequently repeated, a considerable amount of dilatation must be the ultimate result.

All the foregoing means which are of importance in preventing the occurrence of the disease, are practically still more important as preventing its increase when established. Many a man whose wind was merely touched, has become dropsical, &c., by attempts to

renovate himself, by endeavours to climb, hunt, and shoot as he did before his "wind" began to go. Old age has commenced on his chest; he is but between fifty and sixty, and he won't admit the existence of it. He strives against its inevitable consequences, and dies from the effects of the struggle years before he would have done had he shunned the contest.

All measures which oppose the supervention of the degenerative changes of age, are to be sedulously employed, with the hope not only of specially retarding age-degeneration of the lungs and thoracic parietes, but of the body generally.

Diet, carefully regulated exercises, and of drugs, iron and cod-liver oil, especially the former, are among the most potent means for effecting the object in view.

Palliative Treatment.—The distress from which the subjects of Large-lunged Vesicular Emphysema suffer, is due—

1. To shortness of breath;

2. To congestions of distant organs produced mechanically by the impediment to the flow of blood through the pulmonary capillaries;

3. To the abnormities of blood which result from the functional and structural changes of the liver and kidneys especially, consequent on their congestion.

1. The remedies for the shortness of breath vary according to its direct cause. Having regard to treatment, the causes of shortness of breath may be summed up thus:—

(*a*) Organic changes in the walls of the thorax, in the walls of the air-vesicles, and in the capillaries in the walls of the air-vesicles, and dilution of the air received into the air-vesicles at each inspiration, by the excess of air retained in them at the termination of expiration.

Breathing condensed air, it appears probable, temporarily relieves the distress of breathing due to the dilution of the air. Whether it does more than this is doubtful.

(*b*) Catarrhal and other forms of bronchitis.—For the shortness of breath arising from these affections, expectorants which both favour free secretion and expectoration are the great remedies. Ipecacuanha and carbonate and muriate of ammonia, squills and senega are the most potent remedies (see art. Bronchitis).

(*c*) Asthma.—Free secretion and expectoration from the bronchial mucous membrane, affords the most efficient relief in continuous shortness of breath from this complication. Ipecacuanha, squill, ammonia, and senega alone, or combined with sedatives and anti-spasmodics, are the agents best calculated to attain the desired end.

It must not be forgotten that disturbances of the digestive organs, the liver, stomach, and bowels are common in Large-lunged Vesicular Emphysema, and are also frequent exciting causes of attacks of spasmodic asthma in that disease.

(*d*) Congestion of the liver, accumulation of flatus in the stomach and bowels, and loaded bowels by interfering with the descent of the diaphragm, are common causes of shortness of breath.

It is in consequence of this that a full dose of blue pill, or calomel and colocynth, followed by a brisk, warm, saline aperient, so often affords marked relief to the dyspnœa of Large-lunged Vesicular Emphysema.

Blue pill occasionally, aromatic saline antacid aperients, taraxacum with soda, or nitro-hydrochloric acid with aromatics, and attention to diet, are the means best calculated to ward off shortness of breath from these causes.

2. In treating the congestions of organs, two objects have to be kept in view.

1st. To remove the impediment to the flow of blood through the lungs.

2nd. To relieve directly the local congestions.

The impediment to the flow of blood due directly to organic changes in the walls of the air-vesicles and in the pulmonary capillaries, is irremediable. Catarrhal and other forms of acute and chronic bronchitis increase the impediment to the capillary circulation through the lungs; and, therefore, to relieve those affections, is to relieve the congestion of the venous system. Free secretion and expectoration from the bronchial tubes, is the most efficient agent for affording relief in these cases.

Violent cough again impedes the flow of blood through the lungs, and so produces congestion of the venous system.

Sedatives, therefore, by checking cough, become means of relieving local congestions.

Free secretion from the kidneys, liver, and intestinal mucous membrane, relieve the local and general over-distension of the capillaries and veins of those organs, resulting from impediment to the flow of blood through the lungs.

Of diuretics, the ordinary salts of potash, with small quantities of iodide of potassium, are, as a rule, the most efficacious. This class of remedies should be preceded by one or more doses of blue pill, with squill and digitalis.

It is common for diuretics not to act till the tension of the venous system has been, to some extent, taken off by other means. Hence, should diuretics fail when first given, aperients may be employed, and their use be followed by diuretics with advantage.

Blue pill, and other cholagogues, followed by hydragogue aperients, such as cream of tartar with jalap, effect the desired object by promoting a full flow of secretions from the liver and intestines, and so especially relieving congestion of the portal radicles and terminal branches.

A natural relief is occasionally afforded to a congested organ by spontaneous hæmorrhage from it. Cerebral congestion is relieved by epistaxis; congestion of the lungs by hæmoptysis; of the liver and intestines, by hæmorrhoidal bleeding; of the kidneys, by hæmaturia; of the stomach, by hæmatemesis. The blood thus lost may not only relieve the vessels of the organ from which it escapes, but the venous system generally.

When congestion of an organ is extreme, the application of dry-cups, or the removal of a small quantity of blood by cupping-glasses, is sometimes very useful. When the distension of the whole venous system is extreme, the removal of a little blood from the arm gives marked and sudden relief when judiciously performed.

The stomach dyspeptic symptoms are due chiefly to congestion of the stomach following on congestion of the liver. They are best treated by occasional doses of mercurials, saline aperients with mineral acids, and mustard poultices to the epigastric region. These remedies may be followed by small doses of strychnine, and light aromatic bitters.

Aromatics, with alkalies, afford temporary relief to the sense of distension and weight.

3. The congestion of the kidneys is sometimes accompanied by the retention of urinary elements in the blood, congestion of the liver by slight jaundice, and finally by organic diseases of these organs, and then all the abnormities resulting from those diseases follow.

For the special treatment of the conditions of blood dependent on the diseases of the liver and kidneys, see the articles on those diseases.

Here it is only necessary to say that the treatment before recommended for the relief of the congestion of these organs, is that best calculated to secure the removal from the blood of the elements retained in it.

In cases of Atrophous Pulmonary Vesicular Emphysema, the great object is to support the failing general powers. Iron is one of the most important tonics in this class of cases. A moderate supply of stimulants is useful.

When accompanied with profuse purulent expectoration, mineral acids, especially the sulphuric, with small doses of quinine, tincture of the sesquichloride of iron, cod-liver oil, and mild sea-air, are the great remedial agents. Stimulating inhalations are sometimes serviceable.

BIBLIOGRAPHY.—For list of works consulted, see Appendix.

ASTHMA.

By Hyde Salter, M.D., F.R.S.

DEFINITION.—Asthma may be defined as dyspnœa of peculiar urgency and violence, generally paroxysmal and recurrent, often periodic, not necessarily attended by cough or expectoration, accompanied usually by dry râles, and compatible with easy and healthy respiration in the intervals of the attacks.

The History of Asthma may be divided into the History of the Paroxysms and the General History of the Disease.

A. SYMPTOMS OF THE PAROXYSMS.—Before the attack itself sets in it is not at all uncommon for the asthmatic to be aware that it i impending, by certain premonitory symptoms with which his expe rience has made him familiar, and of which it has given him the infallible interpretation. It is, however, more common for there to be no distinct premonitory symptoms, but for the first slight traces of the attack to be the only warning of its approach. When premonitory symptoms *do* occur, they are generally such as are referable to the nervous system ; as, for example, an unusual buoyancy of spirits and mental excitement, or depression, lethargy, and an irresistible sleepi- ness. One very common symptom is profuse diuresis, the patient passing, some hours before the attack, a large quantity of clear pale urine, almost as white as pump-water—identical, in fact, with what is called nervous water, or hysterical water. As a rule, however, as has been mentioned, the patient has no warning, no sign whatever to guide him ; and this is one of the peculiar features of Asthma, and imparts to it that uncertainty and ever-possible nearness which makes it so disqualifying a disease and one so destructive of the engagements of life and the duties of an active career. It always threatens because it *never* threatens. Nay, more, from the time at which it is most.apt to occur—the early morning—the patient has not even the warning of the *initiatory* symptoms, but sleeps through them till he is awoke to find himself with the attack full upon him. He goes to bed every night uncertain whether his next return to consciousness may not be among the full developed horrors of the asthmatic struggle.

The *Initiatory Symptoms*, when they are not slept through, and when they show themselves by day, consist in a faint development of the characteristic dyspnœa of Asthma—a slight sense of constriction across the chest, a short dry cough, a tendency to wheeze, and an

indisposition to exertion. The asthmatic's friends notice that he walks about with his shoulders higher than usual; and he complains of flatulent distension which makes the girth across the epigastrium greater than usual, so that he unbuttons his waistcoat to give himself ease and room. This is not a mere sensation, the circumference of the chest is really considerably increased, and the patient usually attributes it to distension of the stomach with wind. No doubt, in many instances, there *is* a large development of gas in the stomach at the commencement of an asthmatic paroxysm; but in the great majority of instances this increased girth of chest and abdomen is probably due to that enlargement of the cavity of the chest which always accompanies the asthmatic state, and is a part of it, of which more will be said by and by, and which enlarges the girth of the chest by the elevation of the ribs, and of the abdomen by the depression of the diaphragm.

These initiatory symptoms may hang about for some time, even for some days, before they culminate in an attack, the asthmatic creeping about with a gradually increasing sense of constriction across the chest, a more perceptible wheeze and a greater and greater incapacity for exertion. At other times the paroxysm is at once so fully developed that it can hardly be said to have any initiatory symptoms at all.

There is nothing more uniform in Asthma than the time at which the attack is apt to come on. In nineteen cases out of twenty, this is in the early morning, from two to four o'clock. So uniform is this, that it is the exception to find it otherwise. Each case, as a rule, has its own particular time: one will always be awoke at two, another at three; and so unvarying is the time, that the patient often knows exactly what o'clock it is by his asthma waking him. There are, however, two circumstances that vary the time: one, the hour at which the patient goes to bed—the earlier this is, the earlier does the attack come on; and the other, the intensity of the exciting cause—the more powerful this is, the earlier is the attack likely to follow it: thus a supper in a favourable air may bring on the Asthma at three o'clock; in an unfavourable air as early as one. Occasionally, the Asthma will remain in abeyance as long as sleep lasts and develop itself immediately after waking; but this is rare. A not at all uncommon time for the attack to come on is about two hours after dinner. In some, it always appears on getting into bed at night. There cannot be a doubt that the part of the four-and-twenty hours the most free from it is the forenoon, from breakfast to luncheon or early dinner; many asthmatics who suffer more or less at all other times are free then.

When the paroxysm is fully developed, the appearance of the patient is very characteristic. He sits in a fixed position, unable to move, generally leaning forward with his hands or elbows planted on something in order to raise his shoulders; sometimes he *stands*, leaning over some piece of furniture, finding this position easier than sitting: kneeling up in bed, leaning over the pillows, or kneeling on the floor

against a bed or chair, is, in many cases, the easiest position. He is pale, or, if very bad, dusky in complexion; the shoulders are raised, the back rounded, and a sweat often pours off the face from the violence of the respiratory efforts. These efforts are so great that the body is quite convulsed by them, the shoulders are thrown up, the head thrown back, and the mouth opened at each inspiration; and all the muscles mediately or immediately connected with the chest thrown into violent action. The patient cannot bear anything tight around his body, all his clothes must be loosened; all curtains drawn around him, all bystanders crowding about him, seem to increase his sense of suffocation. Sometimes he will sit by an open window the whole night in the coldest weather, so great is the desire for fresh air; he feels as if death was impending, as if his chest were bound with iron, and as if the only thing that would give him relief would be to cut it open. The extremities are often cold, especially the lower extremities, although the perspiration may be running from the face at the same time; the pulse is small and quick.

If the patient is stripped and the chest watched it will be seen that, although the respiratory efforts are so violent, there is very little real movement, the muscles tug at the ribs, but the ribs refuse to rise— they strive to compress them, but they refuse to subside. Although violent the respirations are often not hurried, not exceeding the natural number; but although the number may be natural, in every other respect the respiratory rhythm is disturbed; the inspiration is short and jerky, the expiration inordinately long, often wound up with a sudden pumping out of the last quantities of the expired air; and there is no post-expiratory rest.

On listening to the patient's chest, everything seems lost in loud musical râles of high pitch, and mostly sibilant; among these is also often heard sonorous rhonchus. These sounds are multitudinous, of all pitch, and utterly discordant, squeaking, chirping, mewing, whistling, cooing, snoring, and fifty other sounds. They are almost invariably louder at expiration, sometimes confined to it. The *typical* sounds of Asthma are of this dry character; occasionally, however, moist râles are heard either from the Asthma being complicated with bronchitis, or from the attack approaching its termination, when mucus is being poured out.

But there is one auscultatory phenomenon in Asthma, which, although negative in its character and apt to be overlooked, is far more important and significant than these noisy manifestations of bronchial stricture; it is the almost complete or total absence of the respiratory murmur; this is not only not heard because it is drowned by the other sounds, but because it is really, for the time being, in abeyance; for even when the musical râles are absent, as they sometimes are, the respiratory murmur is equally defective.

The reason of this absence of respiratory murmur is that the bronchial spasm prevents the air reaching the recesses of the lungs in sufficient quantity to generate it. The more severe the Asthma,

the more complete is the loss of the respiratory murmur, and no sooner does the spasm yield than the normal sound is reinstated.

Not unfrequently, the physical signs of Asthma, above described, are partial in their distribution, the respiratory murmur is not totally absent but crops up tolerably clearly here and there; the sibilant and sonorous râles, too, are patchy—some parts of the chest, especially those where the respiratory murmur is best heard, being free from them.

There can, I think, be but one interpretation to this patchy distribution of the physical signs of Asthma; namely, that the Asthma itself has a patchy distribution—that the tubes are in some parts affected, in others free. In those parts where, from the absence of spasm of the tubes, the access of air is free, we often hear a respiratory murmur of strongly-marked compensatory character, the inrush of air remarkably clear and loud. This is just what might be expected, and depends upon the whole of the violent inspiratory efforts being spent upon those parts of the lung where the absence of bronchial spasm renders its inflation possible.

There is yet another fact to be observed; namely, that the physical signs of Asthma change their seat with considerable rapidity: we may hear a patch of sibilus or rhonchus one minute, and the next it may be gone; we may find complete absence of respiratory murmur, and in a quarter of an hour, in the same place, it may be quite audible. In fact, for no two consecutive hours does the asthmatic chest present the same physical signs at the same place. This fugitiveness and migration of the normal and morbid sounds show that the bronchial spasm itself is constantly changing its seat, that constricted tubes are constantly becoming relaxed, and patulous ones contracted. Occasionally, the physical signs show that the Asthma is lateral, confined to one lung, or nearly so, the râles will occupy one lung and normal breath-sound the other. The patient himself is sometimes quite aware that only one of his lungs is affected, and knows which it is. In most of those cases in which Asthma is thus restricted to one side, it always occurs more or less on the same side. In such cases there is probably some organic cause, such as emphysema or bronchitis, for its localization.

Percussion, during a paroxysm of Asthma, is always exaggeratedly resonant; in this, the chest distension, and the loss of respiratory murmur, the conditions bear a striking resemblance to emphysema: in one respect, however, besides the temporary duration of these signs, there is a marked contrast; in emphysema, the intercostal spaces are not depressed, even in strong inspiration, while in Asthma they are drawn in to an extraordinary degree; and not only the intercostal spaces, but all the yielding parts bounding, or lying contiguous to, the chest cavity, as the supra-sternal and supra-clavicular fossæ, and the scrobiculus cordis. The surface in these situations is literally sucked in at each inspiration; and nothing more strikingly suggests than this appearance the real nature of the difficulty in asthmatic breathing,

nothing more strikingly proves that it must depend on a condition which temporarily renders the lung incapable of following the inspiratory enlargement of the chest wall.

Before the paroxysm ceases there is commonly some expectoration. In many cases the fit never ceases without it, and not until the expectoration is established will the spasm give way, however long it may be delayed. It is this circumstance that has given rise to the theory that the material discharged has been the cause of the preceding attack, and that the violent respiratory efforts are merely the mechanism which nature adopts to get rid of it. This was Bree's theory, and expressions frequently used by patients show that such an idea is still very prevalent. They say, "If I could once get the phlegm up, the spasm would give way." No doubt, the phlegm, when there, is a source of irritation and an additional cause of dyspnœa, and no doubt, if it were discharged, the patient would be, *pro tanto*, under better circumstances. No doubt, too, in many cases, the spasm will not give way till the expectoration takes place, but only because the expectoration will not take place till the spasm begins to yield. Till that takes place, the mucus is locked up behind the constricted tubes, and cannot be discharged, partly because its channel of egress is too narrowed, and partly because sufficient air cannot be introduced behind the constricted tubes to produce efficient cough. That the pituitary theory of Asthma—the theory of a material irritant present in the secretion of the tubes—is incorrect, is shown by the fact that there are many cases in which there is no secretion first or last, the chest sounds being dry throughout, and the spasm going off without any expectoration. And even in the cases where the attacks always terminate with more or less spitting, they begin dry. At first there is no cough, and nothing but a dry wheeze; by and by, to the wheeze, rattling is added, and cough begins to appear, and before the paroxysm is over moist sounds may be heard all over the chest, and the cough may be incessant. The mucus has evidently been gradually developed as the attack has progressed. In fact, the spasm is the cause of the secretion, and not the secretion the cause of the spasm. This is not at all inconsistent with the discharge of the secretion, when once formed, being attended with relief.

The material itself is peculiar and very characteristic. True asthmatic sputum, where there is no bronchitis, consists of little pellets of grey pearly mucus, like pieces of tapioca, or very firm arrowroot. It is free from pus, free from either stringy or watery mucus and not frothy.

Rarely, another material is expectorated during the attack—blood. In most instances where this occurs it is only characteristic of the severest paroxysms; in some few cases I have known every attack attended with more or less blood-spitting. It is generally small in quantity, in streaks and patches; sometimes it amounts to a profuse hæmorrhage. It evidently depends upon the rupture of the overdistended bronchial venules and capillaries, due to the congestion into

which they are thrown by the partial asphyxia of the asthmatic paroxysm.

The *duration* of a fit of asthma is a thing about which there is no rule—it may be over in a few minutes, it may last many weeks. But though there is no rule for the *disease* there generally is for the *case;* each case has, as a rule, its own length of attack although the uniformity may not be rigid. A very common length is for the attack to begin at three or four o'clock in the morning, and be over by breakfast time, or gradually clear off at nine or ten o'clock in the morning. In many cases, the attack involves a single day, never more or less. From two to three days is not an uncommon duration. In some cases, the duration is complex, each attack extending over many days and consisting of a succession of shorter paroxysms with easy breathing between; and then, the bout being over, many months may be passed before another attack occurs. This is especially the case in those instances in which the asthma is due to some cause that occurs at distant intervals, such as hay Asthma. Here the asthmatic state will last, more or less, throughout the whole of the grass-flowering season, although consisting throughout that time of short paroxysms, each not lasting above an hour or so, and perhaps confined to the night.

The *method of termination* of an attack depends very much upon two circumstances : one, the length of time the attack has lasted ; and the other, whether it yields spontaneously, or in obedience to remedies. If the attack has lasted long it is always slower and more protracted in its departure ; for the lungs are left so congested that it may be days before their circulation can resume the condition it was in before the fit. For the same reason, the expectoration after a prolonged attack is more profuse and continues longer. Again, if an attack is left to die out by itself, its departure is often tedious, and it may show many partial remissions before it takes its final leave. If, however, some powerful influence is brought to bear upon it, it may yield almost instantaneously—the patient may be one minute struggling for breath and the next without a trace of dyspnœa, as for example where the Asthma is suddenly arrested by some violent emotion, as fear, or where it yields to the influence of some powerful depressant, as tobacco. Unless the paroxysm has been of very short duration it generally leaves the patient with a sense of clogging and stiffness at the chest; he feels himself more than usually incapable of exertion, and is easily winded : this gradually gets less and less, and in a day or two may have completely passed away. The expectoration often continues for several days after the attack is quite gone; at first, it occurs throughout the day, is then confined to the morning, and finally ceases altogether.

There is one peculiarity in the state of the asthmatic after the paroxysm that is especially worthy of remark, and that is the diminution of the asthmatic tendency that he then experiences—the almost certain immunity, for the time being, from a repetition of the attack. There are many things that he dare not do at other times without the

certainty of bringing on a paroxysm, that immediately after an attack he may do with perfect impunity. It seems as if each fit were a sort of "clearing shower," as if the tendency to fall into the asthmatic state accumulated in the intervals, and was, so to speak, discharged by the paroxysms. Certainly the fact, which we frequently see in Asthma, that the longer the time that has elapsed since the last attack the more particular must the asthmatic be in not exposing himself to the ordinary exciting causes of his disease, and the more sensitive of their influence does he become, is compatible with this idea. We see just the same thing in epilepsy.

B. Such is the history of an asthmatic paroxysm. But, besides the features of the paroxysm, there are certain points in the history of the disease that deserve notice. Of these I will especially advert to three :— First, the periodicity that the disease so frequently exhibits ; secondly, the change of phase that time impresses on many cases ; and thirdly, the influence which sex and age appear to exercise on the liability to the disorder.

1. *Periodicity.*—That Asthma is markedly periodic no one who has watched it can doubt. Although in some instances there appears to be no particular interval at which the attacks are apt to occur, yet, in the majority of cases, the interval is well marked, and in many, minutely and singularly regular. This is one point, among many others, that vindicates for Asthma a place among the neuroses. But while each case preserves its own periodicity with more or less regularity, there is no particular period for *Asthma* itself ; for, while in one case the attacks will occur regularly at the same time every night, in another they will occur once a month, in another once a year. So regular is the time of recurrence in some cases that the asthmatic knows exactly when to expect an attack. The periodicity of Asthma is clearly divisible into two kinds, *intrinsic* and *extrinsic.* Of the former, which is the only true essential periodicity, we see examples in those cases in which the attack comes on after the same interval irrespective of external circumstances. Of the latter we see examples in those cases where the regular return of the attack is simply due to the regular return of the exciting cause. The period in the last cases is almost always that of some natural interval. Thus the annual periodicity of hay fever is of this kind ; so is the monthly periodicity of hysterical Asthma, and the diurnal periodicity of cases in which sleep and the recumbent posture induce the attacks. Indeed, in any case in which the periodicity affects some natural interval I should suspect that it was extrinsic, and dependent on the periodic recurrence of the exciting cause. Thus, in all cases in which the attack comes on every Saturday, or every Sunday, or every Monday morning, I believe the attack is due to something having a weekly recurrence, something in which Saturday and Sunday differ from other days—a suspension of the usual employment ; difference of food, or the time of taking it ; sleep after food ; the taking of supper. That the periodicity of such cases is not inherent

or essential is shown by the fact, that if the exciting cause is made to recur at irregular intervals the attacks become correspondingly irregular, and all periodicity is lost.

2. *Change of Type by Time.*—It is not at all uncommon to see the features of a case change considerably as time advances, and ultimately differ very much from what they were at first. And there is a certain type of change that commonly obtains, so that it is possible in any given case to predict what the effect of time will probably be. As a rule, the attacks are the most violent in the early history of a case, and gradually become less and less severe. It is very common for patients to say, "I never have those awful attacks now that I used to have, they seem to have quite left me." But while the attacks become milder they often become more frequent, so that a monthly periodicity may be exchanged for a weekly, or a diurnal one. At the same time, another change is generally going on—the breathing in the interval is getting less and less free ; certain slow organic changes are gradually being impressed on the lungs by the repetition of the attacks, by which their functional integrity is increasingly impaired ; so that while at first the attacks are severe and distant, and the breathing in the interval like that of a healthy person, after a time the paroxysms become so slight and frequent, and the breathing so embarrassed, even at its best, that there can hardly be said to be any distinct attacks, and the disease has ceased to be paroxysmal.

3. *Age* and *Sex.*—It is a commonly received opinion that Asthma is a disease of advanced life. Nothing can be more erroneous. It is confined to no age ; and so far is it from being peculiarly a disease of the old, that I find a larger number of cases take their origin in the first ten years of life than in any subsequent equal period.

During youth, from ten to twenty, few cases originate ; but, from that time up to fifty, the asthmatic tendency regularly increases. From that time forward, fewer and fewer cases take their origin. No doubt, many old people are asthmatic; but that is simply because many asthmatic people reach old age. It must be borne in mind, too, that with respect to old people the word "asthma" is very loosely used : three-fourths of the "asthma" of old people are due to chronic bronchitis.

Men are liable to Asthma, in relation to women, in the proportion of two to one.

C. Varieties of Asthma.—All cases of Asthma fall, I think, under one of two main divisions—Idiopathic or Primary, and Symptomatic or Secondary. Idiopathic Asthma is the inherent and essential form of the disease that occurs independent of and uncomplicated with any other affection. The best marked, most typical, and characteristic cases, and I may add the most severe, are of this kind. In these cases we generally get considerable intervals between the attacks, those intervals being marked by perfect freedom of breathing, and the attacks by a regular periodicity. There are many points of resemblance between this variety of Asthma and epilepsy. Both of them affect

the same nervous temperament, both of them are markedly periodic, and in both each paroxysm seems to act as a sort of thunderstorm, and to discharge, or work off, some particular state which constitutes the liability to the condition, and which accumulates in the intervals, and reaches its maximum immediately before the fit. So close a relation, indeed, exists between this form of Asthma and epilepsy, that I have seen two or three well-marked cases in which the one kind of fit took the place of the other. Most examples of Asthma in the young are of this idiopathic type.

Symptomatic or Secondary Asthma may be subdivided into three varieties—peptic, bronchitic, and cardiac—that is to say, Asthma may have its origin in stomach derangement, in an inflamed condition of the bronchial mucous membrane, and in heart disease. Each of these varieties has something peculiar in itself, depending generally on the nature of its cause. Thus *peptic* Asthma is apt to come on two or three hours after taking food, may be entirely regulated by dietetic rules—brought on at any time, or kept off indefinitely at pleasure—according to what is eaten, and when. It is remarkably independent of the other recognised causes of Asthma, and is (through the stomach) more amenable to treatment, and more hopeful, than any other form. Bronchitic Asthma, perhaps the commonest of all, is distinguished from all other varieties by certain well-marked characteristics. It is only caused by the causes of bronchitis, especially cold. As a rule, the patient never has Asthma without bronchitis, and never has bronchitis without Asthma, so that we generally have really a complex condition to deal with, although the bronchitic element may sometimes be so slight as hardly to be detected. There is generally in these cases an abundant expectoration and a good deal of cough and moist breath sounds. Such cases are often very intractable, and from this reason, that we have two diseases to treat— bronchitis and Asthma ; the bronchitis is intractable because it is so greatly aggravated by the Asthma, and the Asthma is intractable because its exciting cause, the bronchitis, abiding, any remedies that are brought to bear upon it are rendered inoperative or merely of transient efficacy. Indeed it so happens, that in one element of treatment, air, that which is best for the bronchitis is often worst for the Asthma, and *vice versâ*. Thus many of these cases lose their asthmatic tendency in London, where the bronchitis alone survives ; while if you send them to the Mediterranean for the cure of the bronchitis, the asthmatic tendency is so much increased, that they are worse than ever, so that they have the alternative of bronchitis at home or Asthma abroad. Being dependent on the causes of bronchitis, such cases are generally worse in the winter ; indeed, in many of them, the Asthma occurs *only* in the winter. I think most of the cases in which Asthma occurs every morning are of the bronchitic kind, the reason being that the inflamed condition of the bronchial mucous membrane constitutes an ever-present exciting cause, which, in a person with the asthmatic tendency, only

requires sleep and the recumbent posture in order to bring it into activity. The third variety, the least common of all, is Cardiac Asthma, or Asthma complicating heart cases, and depending upon the heart disease. A great deal that goes by the name of Cardiac Asthma is not Asthma at all; it is simply cardiac dyspnœa, un-attended with any bronchial spasm. Now and then, however, a heart case is met with in which paroxysms of true Asthma occur, attended with wheezing, prolonged expiration, and other characteristic signs of Asthma. In these cases, I have no doubt that the immediate exciting cause of the Asthma is the pulmonary congestion produced by the heart disease.

This last variety, and bronchitic Asthma, when the bronchitis has become chronic, may be classed together as " organic " Asthma; the peptic variety and the idiopathic, as non-organic.

D. CAUSES OF ASTHMA.—The causes of Asthma may be divided into two classes—those affecting the air-tubes primarily and directly, and those applied to some remote part. Of those that are brought to bear directly upon the air-tubes there are three kinds: first, things inhaled; secondly, some offending condition of the blood; and, thirdly, an in-flamed condition of the mucous membrane of the air-tubes.

There is an endless variety of materials which, when respired, will produce Asthma in those possessing the asthmatic tendency, and they produce it no doubt by virtue of that morbid sensitiveness of the bronchial mucous membrane in which the Asthma in these cases essentially consists. Some of these materials are such as will produce a certain amount of Asthma in many people, such as the smell of a lucifer-match, pitch, smoke, pungent vapours. Some are rendered asthmatic by dust, some by fog and damp. Particular smells will at once bring on Asthma in some people, such as that of flowers—roses, for example, and privet. The commonest vegetable emanation having this effect is hay—this form of Asthma being well known as Hay Asthma, and a part of that curious disease, Hay Fever. In some people animal emanations have a similar effect: some are at once rendered asthmatic by the presence of a cat, some cannot go near a stable, or even ride behind a horse, or go near those who have been riding; some the effluvium of rabbits renders asthmatic; some, guinea-pigs; some cannot go near a poulterer's shop where there are hareskins; some have their Asthma brought on immediately if they go to a menagerie; and some suffer immediately if a dog comes near them. A more subtle influence is that arising from change of weather, or particular winds, some persons being rendered at once asthmatic by an easterly wind. A more subtle influence still is that arising from locality. Almost all asthmatics are influenced to a certain degree by the air they breathe, but to many it is the one thing that regulates their Asthma. Some are best in a dry air, some in a moist, some high, some low, some inland, some by the seaside. In some there is only one place that will render them asthmatic, in others there is only one

place at which they are free from Asthma; in some the peculiar character of air that offends is well known; in some it is utterly inscrutable. In some so slight is the peculiarity of air that will determine the supervention of Asthma, that they may be perfectly well in the front of the house, but cannot sleep at the back. As a rule, a dry air is worse for Asthma than a rather moist one, the air of a high locality than a low. Yet the most constant circumstance noticed in respect to air, is the superiority of urban air over that of the country. So common an incident is this, that it becomes an important element in treatment—many a case of Asthma is at once cured by living in a dense quarter of some smoky and crowded city.

The cases in which I am inclined to think Asthma is brought on by *an offending condition of blood,* are cases in which it is apt to come on a little time after the ingestion of certain articles of diet. Cases in which people are asthmatic about two hours after a meal (a very common circumstance) are of this kind. In some only certain articles of diet will give rise to the Asthma, as wine, beer, sweets; in some only what upsets the digestion; in some any food whatever. My reason for thinking that it is the condition of the blood circulating in the respiratory organs after the absorption of these ingesta, that produces the bronchial spasm, and not the irritation caused by the presence of food in the stomach acting on the gastric periphery of the pneumogastric nerve, is the time at which the Asthma comes on. If it were the presence of the food in the stomach that caused the Asthma the symptoms would appear immediately on taking it, whereas it is not until a couple of hours afterwards, at about the time when the results of digestion are entering the circulation, that the difficulty of breathing comes on. Moreover, the rapidity with which the Asthma will supervene varies as the rapidity with which the ingesta are absorbed; thus, after wine, which is rapidly taken up, the Asthma will very quickly make its appearance.

Inflammation of the bronchial mucous membrane is one of the commonest causes of Asthma, perhaps the commonest of all, especially in people in advanced life. Such cases are really complex cases, being bronchitic as well as asthmatic; indeed the bronchitic may be said to be the fundamental and essential part of them, and their essential treatment is the treatment of the bronchitis. Take care of that and the Asthma will take care of itself. The only difference between such cases and cases of ordinary bronchitis, is that the bronchitis happens to occur in individuals in whom bronchial spasm is easily induced.

The immediate excitants of the asthmatic paroxysm to which I have already referred are such as act directly on the bronchial tubes; but there are some that produce bronchial spasm by application to some remote part. Such causes always act, I believe, through the nervous system; and they may either act through the organic nerves or the cerebro-spinal. We see an example of the former in cases where Asthma is at once produced by a loaded stomach, or a loaded rectum: of the latter, where Asthma is at once produced by cold

feet, &c. In both these classes of cases the exciting cause is applied to the periphery of the nerves on which it acts; but this need not be the case, for, sometimes, the irritant is applied to a nervous centre. Asthma, for example, has been known to be produced by organic disease of the brain; and that very common occurrence, the production of Asthma by violent emotion, is another example of the same thing; only here the irritant applied to the centre is psychical and not physical.

In speaking of the causes of Asthma I must not omit to mention those which lay the foundation of the asthmatic tendency. Perhaps the largest group of causes of this kind are conditions affecting the vascularity of the bronchial tubes, such as measles, hooping-cough, bronchitis. In many cases the sole predisposing cause appears to be some inherited peculiarity.

E. DIAGNOSIS OF ASTHMA.—It is of the utmost importance to be able to recognise Asthma with certainty, because there are several diseases with which it might be, and often is, confounded, and because the treatment of these diseases and of Asthma is of the most opposite kind.

The three forms of dyspnœa with which Asthma is apt to be confounded, are bronchitis, emphysema, and heart disease. From bronchitis, Asthma may be distinguished by its sudden access, and often equally sudden departure, by the absence of cold as a necessary cause, and frequently by the absence of expectoration and of moist sounds. Moreover, when expectoration does occur, it is of a different kind; in bronchitis it is often purulent, in pure Asthma never. Again, the action of remedies distinguishes the two dyspnœas: the intensest asthmatic dyspnœa will often suddenly, almost instantaneously, yield to certain remedies; in bronchitis this is not the case; if the dyspnœa is severe, so as to be at all commensurate with Asthma, it always takes some time to subside.

From emphysema, Asthma may be distinguished by the paroxysmal character of the dyspnœa, by its violence, and by the absence of any dyspnœa whatever in the intervals. In emphysema, the cause of the difficulty of breathing is organic and unchanging, and, therefore, dyspnœa is never completely absent, and varies in amount only in proportion to the degree to which respiration is taxed. The presence or absence of the physical signs of emphysema will also, of course, materially aid the diagnosis.

The dyspnœa that Asthma is the most apt to be confounded with, and which it most resembles, is that of heart disease. The two resemble one another in that they are both paroxysmal, both intense, both apt to occur at night, both compatible with organic soundness of lung, and both intolerant, though not exactly in the same way, of the recumbent position, of exertion, and of sleep; moreover, in both of them the respiration may be perfectly normal between the attacks. It is not wonderful, therefore, that with so many points of

resemblance, the two should sometimes be confounded. There is, however, no real difficulty in distinguishing them. In cardiac dyspnœa there is generally an absence of the characteristic signs of narrowing of the bronchial tubes, universally present in Asthma, such as wheezing, prolonged expiration, suppression of respiratory murmur, &c. The length of the attacks, too, is different, the asthmatic paroxysm being commonly longer than the time reached by an attack of cardiac dyspnœa.

F. Prognosis of Asthma.—This varies greatly in different cases, in some being unqualifiedly favourable, in some unqualifiedly unfavourable, and in some doubtful; it is principally influenced by the following considerations :—

1. *The presence or absence of an organic cause.*—If the Asthma is manifestly dependent on some organic cause, in its nature irremediable and irremovable, it is manifest that the resulting Asthma must be itself incurable. If, for example, it depends upon inveterate bronchitis it is clear that all treatment can be merely palliative, and that a final cessation of the Asthma can never be expected. If, on the other hand, the circulatory and respiratory organs are found to be perfectly sound, then, *quoad* this circumstance, the prognosis is favourable ; for, though the absence of organic disease does not make the final cessation of the Asthma certain, it makes it possible; in other words, the absence of organic disease makes the prognosis negatively favourable though not positively so.

2. *Age* has great sway in influencing the prognosis of Asthma ; the younger the individual the more probable is ultimate recovery : an asthmatic child of ten will probably lose his Asthma, an asthmatic man of forty will probably not; an asthmatic man of sixty you may say, will certainly not ; in an asthmatic youth of twenty it would be difficult to say, as far as the circumstance of age goes, on which side the probabilities would lead. The reason for this fact appears to be, that Asthma has much more commonly an organic basis in advanced life than in early life, that the tendency of Asthma to lay the foundation of organic change is much greater in advanced life than in early life, that the loss of a constitutional peculiarity is much less probable in advanced life than in early life.

3. *The frequency and severity of the attacks* very much influence the prognosis ; for if the attacks are very severe and very frequent the lungs are unable to recover in the intervals from the injuries inflicted by the attacks, and certain organic changes are probably and speedily induced. If, on the other hand, the attacks are light, and the intervals between them long, the lungs are able perfectly to recover from the temporary derangement produced in them by the paroxysms ; and such a case may go on for an indefinite time without the development of any organic changes.

4. The state of the patient in the intervals is of great importance in influencing our views as to prognosis. If the lungs and heart

appear to be anatomically and functionally sound, if the breathing is perfectly natural and free, and there is no wheezing, cough, or expectoration in the intervals, the prognosis is infinitely more favourable than if the reverse is the case. Persistent difficulty of breathing in the intervals of the attacks is a very bad prognostic sign; indeed, I think the state of the respiration in the intervals of the attacks is of more importance than either their frequency or severity.

5. Lastly, the history of the case often greatly influences our prognosis, because the past often implies the future; if we find that the tendency of the case—the direction it appears to be taking—is towards an alleviation of the symptoms, the attacks becoming lighter or less frequent, or in any way mitigated, we have strong warrant for a favourable prognosis; if, on the other hand, the attacks have been becoming more frequent, more easily induced, more violent, or protracted, or in any way aggravated, then a favourable issue becomes exceedingly improbable.

G. PATHOLOGY OF ASTHMA.— Our views respecting this must be greatly influenced by our views of the immediate condition in Asthma. My belief is that the immediate and essential condition of the asthmatic paroxysm is a state of contraction of the bronchial tubes. What proof have we of this? In the first place, the sudden induction and remission of the asthmatic paroxysm is consistent with its depending on muscular spasm; in the second place, there is abundant proof that the air in the lungs is locked up, and can neither be got in nor out: there is evidently plenty of air in the chest, percussion is even hyper-resonant; the patient is as unable to drive air out as to draw it in, can neither inspire nor expire, cannot discharge breath enough to whistle or blow out a candle, or blow his nose. The muscles of respiration tug and labour to fill and empty the chest, but the chest walls remain almost immovable: the inspiratory muscles cannot raise them, the expiratory cannot depress them. On listening to the chest we find corroborative evidence of the stagnation of the air. The respiratory murmur is in a great degree lost. This absence of respiratory sound, accompanied by violent respiratory effort, is one of the most striking and suggestive of the facts of Asthma. How can we explain it, except by supposing that there is some bar to the ingress and egress of air; and what can this bar be, unless it is spasm of the bronchial tubes? It cannot be inflammatory thickening of the mucous membrane lining them; for the sudden, almost instantaneous, establishment and remission of the dyspnœa is incompatible with this. It cannot be mucous plugging of the tubes; for the attack will often come and go without any expectoration whatever. But we have still more positive and precise evidence of circumscribed narrowing of the air-tubes in the musical sounds that are present in asthmatic breathing. This symptom has all the certainty and precision that characterise physical phenomena, and shows that the air-tubes are the seat of constrictions that throw the air passing through them into vibrations, and convert them into musical

instruments; and since these musical sounds are multitudinous the points of constriction must be many; and since they are constantly varying in locality and character, the constrictions of the tubes must be undergoing similar change. Lastly, the effects of remedies and their nature tell the same tale, and point to muscular spasm as the immediate essential condition. The most powerful remedies of Asthma are what are called cerebro-spinal depressants, such as emetics, tobacco, &c.—remedies whose direct effect is to relax muscular spasm.

If, then, the immediate condition is muscular spasm, the presumption is, that the primary and essential condition is an affection of the nervous system; with very few exceptions we may lay it down as a rule that perturbed muscular action points not to the muscular system, but to the nervous. What proof have we, then, that the nervous system is involved in Asthma? Some of the most striking proofs of this are derived from the nature of the causes of Asthma; and many of these not only show that the nervous system is the real seat of the morbid action, but they show also what portion of the nervous system is involved. The most numerous of the causes of Asthma are what may be called respired irritants, noxious materials of whatever nature contained in the inspired air. It is manifest that these can only be appreciated by the perceptive nerves distributed to the bronchial mucous membrane, and that they can only give rise to bronchial spasm by the irritation which they produce being propagated to the bronchial ganglia, and by them reflected to the motor filaments distributed to the muscular wall of the bronchial tubes. This, then, is the nervous circuit involved in these cases—extremely short, but still a distinct nervous circuit. In other cases an undigested meal will produce Asthma; here the nervous circuit is longer, and involves the gastric branches of the vagus as its afferent portion, and the pulmonary as its efferent. In other cases a loaded rectum, or uterine irritation, may be the cause of the paroxysm; here the circuit is still longer. In other cases, the sudden application of cold to the surface may at once induce bronchial spasm; here the circuit involves the cerebro-spinal as well as the ganglionic system of nerves. In other cases, some sudden emotion may at once throw the patient into a paroxysm of Asthma; here there is no true circuit, no reflexion, but the stimulus is propagated direct from the centre to the periphery.

There are other circumstances that point equally clearly to the nervous nature of Asthma. The action of many remedies is not explicable on any other hypothesis. For example, emotion will not only cause Asthma, but it will cure it, and in the most sudden and complete way. Nervous stimulants, such as coffee, strong forms of alcohol, &c., are very powerful remedies; and nervous sedatives, such as stramonium, are among the best known and most efficient of our means of relief.

The view that the nervous system is essentially engaged in the asthmatic state, does not negative the fact that the foundation of Asthma may be laid, and the asthmatic tendency determined, by something organically affecting the respiratory organs. All who are familiar with

Asthma must have observed that it frequently takes its origin in child-hood from measles, or hooping-cough, or bronchitis. Now, these are diseases disturbing the vascular condition of the bronchial mucous membrane ; but a morbidly vascular mucous membrane is a morbidly sensitive mucous membrane, and, therefore, a mucous membrane whose irritation is likely to produce, through the nerves supplied to it, spasm of the muscular wall of the tube which it lines, just as we see spas-modic stricture of the urethra apt to occur in gonorrhœa.

H. Treatment of Asthma.—There are two things that the physician has to do—two problems suggested to him—in the treatment of any case of Asthma : one is to relieve the attacks when they occur, and the other is to prevent their occurrence ; in other words, one is the treat-ment of the paroxysm, and the other the treatment of the disease. Of these two the latter alone deserves the name of curative treatment, the former is merely palliative. I shall first consider the treatment of the paroxysms.

On being summoned to a patient in an asthmatic paroxysm, the first thing that the physician has to do, is to remove any exciting cause, and to place the patient in the most favourable condition. If an offending meal or some error in diet appears to be the exciting cause, an emetic should be at once given ; if a loaded rectum, a purgative should be administered, &c. ; if smoke or dust, or any vegetable or animal emanation, is the cause of the attack, this cause should be immediately removed ; free ventilation should be secured, and the crowding and officious ministrations of friends should be forbidden. The sufferer should not be made to speak ; everything should be done for him, and done without the necessity of his requesting it. The position in which he is placed will make a great difference to him, not only to his com-fort, but to the abatement of his symptoms. The best position to put the asthmatic in, as a rule, is sitting in a chair and leaning forward on something in front of him, so as to raise his shoulders. Sometimes he will find leaning on something in a standing posture the easiest posi-tion ; but, generally, standing involves too much exertion. Sitting at a table and leaning forward so as to rest the elbows on it, or resting the elbows on the arms of an arm-chair, or kneeling up in bed, or kneeling on the floor and resting the elbows on the side of the bed or a chair, are positions that give the greatest relief.

I have known one patient who elevated the shoulders by placing under them two short crutches which rested on the side of her chair. The great object is, in some way or other, to raise the shoulders, and the advantage of doing so by these mechanical means is that it saves the muscles the labour and fatigue of so doing. The reason why it is necessary that the shoulders be in some way raised, is that the inspi-ratory muscles passing from the shoulders down to the chest wall may act with greater power as elevators of the ribs.

Having then placed his patient under the most favourable circum-stances for the abatement of the spasm, the physician has next to

select the remedies that he will employ. This selection is very much influenced by the patient's experience. From the constitutional nature of Asthma, and its persistent character, it is rare to see a patient in his first attack. The great majority of asthmatics that one sees are habitual sufferers from their disease, and have generally some knowledge, often a very accurate one, of the remedies that best suit their case. But in this there is the greatest variety, and the experience of one person would be no guide to the treatment of another. Indeed, the behaviour of Asthma to remedies is marked by the most extraordinary uncertainty and caprice; that which is the most valuable in one case is inert in another; in some there are many things that will give relief,—the only question being which is the quickest and the most complete; in other cases all remedies are alike powerless.

The remedies of the asthmatic paroxysm may, I think, be divided into three classes :—Direct Depressants, Sedatives, and Stimulants.

Depressants.—I have already spoken of the value of emetics for the purpose of evacuating the stomach of unwholesome or undigested contents. No doubt, in this way, by the removal of an exciting cause, the paroxysm may often be relieved. But emetics also relieve Asthma very efficiently as depressants, quite independently of their emetic action. I may be asked, " How do I know that it is as depressants, and not as evacuants of the stomach, that emetics give relief?" For two reasons: first, because they will give the same relief when the patient has an empty stomach; and secondly, because the relief comes on when the first sense of nausea is experienced, and before any vomiting has taken place; in a moment, at the first sensation of faint sickness which gives warning of the approach of vomiting, the spasm will suddenly yield, and the patient pass into a state of tranquillity and ease. If this condition could be produced and kept up without giving rise to vomiting I think it would be just as well for the asthmatic, provided that the paroxysm was not kept up by a loaded stomach. The emetic that I have most commonly given is ipecacuanha powder, in twenty-grain doses; it generally acts in a quarter of an hour; a tumbler of warm water should be taken before its first action and after each act of vomiting. I have lately thought the ipecacuanha wine preferable to the powder, from its action being sooner over; the powder, I think, sometimes sticks to the surface of the stomach, and keeps up a teasing and lingering retching. With a view to produce nausea, short of vomiting, I sometimes give ipecacuanha lozenges, directing the patient to take one at short intervals till a slight sense of nausea is experienced, and to return to the lozenges as soon as this passes off; this plan is often quite successful.

Tobacco.—As this is one of the most powerful depressants, so it is one of the most powerful remedies of Asthma. In those unaccustomed to its use, and in whom, therefore, its full physiological effects are most developed, it is almost impossible for the asthmatic paroxysm to resist it. If I were asked to name a remedy on which I should place the greatest reliance in subduing the most obstinate asthmatic

spasm, I should say tobacco in those unaccustomed to it. I believe that the death-like collapse that it produces is something before which Asthma must go down. From this potency it is in obstinate cases a most valuable remedy, but it has three disadvantages : in the first place, it is peculiarly distressing—the sensation that it produces is as near like the worst form of sea-sickness as possible, perhaps a little worse; in the second place, it sometimes produces alarming, if not dangerous symptoms ; and in the third place, it is, in a large class of asthmatic patients, adult males, inoperative in consequence of its habitual use.

It should always be given with great care, and tentatively, especially to those who have never before tried it ; and the mildest forms should be chosen. Like ipecacuanha, tobacco relieves Asthma independently of the vomiting it may produce. By careful management and experience, smoking may be carried just far enough to give rise to a sense of faintness and slight nausea, without its passing on to vomiting at all.

Another remedy, very efficacious, very commonly used, and very like tobacco in its action, is the *Lobelia inflata*. I find that different authorities have a very different estimate of the value of this remedy ; and I myself am conscious that I have a much higher opinion of it than I had some years ago. The fact is, I now give it in a way that I believe tests its powers much more fairly than the ordinary way in which it is administered; this is the plan recommended by Dr. Elliotson, of giving it in gradually increasing doses at short intervals, till its physiological effects are manifested. I generally start with twenty minims of the æthereal tincture, and tell the patient to repeat the dose every half-hour, making it five minims larger each time, till some slight nausea and feeling of faintness is experienced. By this plan the efficacy of the drug is fairly tested ; by the ordinary plan of giving a patient fifteen or twenty drops every three or four hours, its value is not tested. I have on many occasions known as much as forty or fifty drops reached before any feeling of nausea was produced, and before the Asthma was relieved ; but with the nausea came the relief. I should never feel the slightest confidence that Lobelia was valueless as a remedy, in any given case in which it had been administered in the ordinary way. I have many times, by changing the method of its administration, obliged patients to reverse their verdict of it. When a patient has found out his maximum dose, I advise him on the next occasion to start with that dose ; it saves him the trouble and loss of time of gradually working up to it.

Sedatives.—The relief obtained in Asthma from this class of remedies, no doubt depends on their rendering the nervous system less irritable and less susceptible to sources of disturbance, and the presence of sources of irritation less likely, therefore, to issue in the production of spasm. Some of them appear to act locally, on the nervous system of the lungs alone, but most of them on the general nervous system. Those that experience has shown to have the most

value in Asthma, are : tobacco, in sedative doses, stramonium, datura tatula, belladonna, conium, hyoscyamus, æther, and chloroform; and lastly, the fumes of burning nitre paper may be mentioned in the same category.

Tobacco, smoked in the ordinary way, is certainly of great service to many asthmatics. By its habitual use, they keep themselves much freer from attacks than they are without it, and are constantly able to check the asthmatic tendency when it shows itself. If at any time a little wheezy they resort to their pipe or cigar, and soon experience its soothing effects; the breathing quiets down and becomes clear, and they are soon themselves again. Many asthmatics have told me that they are sure that, if they left off smoking, their asthma would soon become troublesome; and that, as long as they smoked, they may do many things with impunity that, without their tobacco, would be sure to bring on their symptoms. But, while very useful in this way, I do not think that it is equal to the subduing of a severe attack, unless pushed to what may be called a poison dose; and then it ceases to be a sedative and becomes a depressant.

The two species of datura—the *D. stramonium* and the *D. tatula*—certainly deserve a very high place among the remedies of Asthma; they are however of very variable efficacy in different cases; and that is the probable reason why different observers entertain such different opinions of their value : some thinking very highly of them and some regarding them as next to worthless. I find, in my own practice, that in the majority of cases they do some good, and in many are the one sovereign remedy. I have had some cases that I may say have been completely cured by them, and others in which, though they have not effected a final cure, the disease has, under their continual use, lost all its horrors. It does not, however, do to speak of them together as if their operation was always alike. In most cases they differ in their effects; in some, one being the most powerful, in some the other. In some cases one will be completely successful, while the other is perfectly inert. Seeing that they not only belong to the same class of remedies, but are merely different species of the same genus, this diversity of their action is very extraordinary. Of the two, I think the tatula is the more powerful. I have, however, met with many cases in which it has been powerless where the stramonium has always given relief. They may be given in two ways,—either by smoking the leaves in a pipe or cigar, or else internally as tincture, or extract; though I doubt if in these two ways exactly the same agent is given. I doubt whether the combustion in smoking does not produce something that did not before exist, as in the case of tobacco-smoking. Nevertheless it is certain that in both ways the daturas are of value. When smoked they are best used with one of two objects, or both—either habitually, at stated intervals,—say night and morning,—with a view of keeping off the attacks and making them less likely to come on; or having them always in readiness to fly to on

the least approach of an attack, so as to check it at once and prevent its development. This latter plan often answers very well. The patient fills his pipe and puts it by the side of his bed over-night, with the means of lighting it, and when he wakes towards morning with the first traces of his Asthma upon him, he at once lights it and smokes away, the dyspnœa subsiding each whiff that he draws; so that in a few minutes he is able to put it out, and lie down and go to sleep again. This is the story of many asthmatics, and they would rather not go to bed at all, than do so without their stramonium by their side. Internally I often give, sometimes with advantage, the extract of stramonium in a pill. I give it in a quarter of a grain dose generally, combined with an eighth of a grain of belladonna, and two or three grains of extract of conium. This pill, taken at bed-time, has sometimes the effect of preventing the development of the attack during the night; it guards the patient through the critical time, and tides him over it. But I must say that I think stramonium taken internally has not that general utility that it has when smoked, and I have known it quite useless in patients, who, when they have smoked it, have found it very efficacious.

Conium and *hyoscyamus* are sedatives that doubtless have some value in Asthma, and are very commonly employed; but, in the majority of cases, the relief they give is but slight and temporary, and they are not remedies on which reliance can be placed. I think I have seen them most efficacious when given in combination, in the form of tincture, with chloric æther. Of *belladonna* I have made an extensive trial since this article was first written, and I am satisfied of its great value in many cases. In not a few its employment has resulted in a complete and apparently permanent cure. I generally give it in the form of the tincture at bed-time, increasing the dose each night, until the asthmatic tendency ceases to show itself, or until, without such result, the physiological effects of the drug are well marked.

Chloroform.—There is perhaps no disease in which the wonderful power of chloroform is more shown than in Asthma. I have never seen a spasm that it failed to subdue. The worst of it is that its operation is often evanescent;—as soon as its physiological effects pass off its remedial effects disappear too. This, however, is by no means always the case, the cure frequently remaining permanent after the stupefying effects of the agent have quite passed off. And even where the Asthma does return, it is no slight thing to be able to suspend its horrors for a time, and to give the sufferer a short respite. It has certain disadvantages that would induce me not to place it among the first remedies that I would try, but to keep it rather as a last resort when everything else has failed:—in the first place, it is, as we know, not entirely devoid of danger; in the second place, it is often not safe to trust it in the patient's hands or those of his friends, and, therefore, can only be used in the presence of the medical attendant; in the third place, its habitual use is very apt to generate a liking for it, and

to pass into a kind of dram-taking. I have seen two or three painful cases of this kind that make me always unwilling to begin its use, just as I am unwilling to begin the habitual use of opium in any chronic malady. In those rare cases in which Asthma never comes on during sleep it is of great value as inducing sleep. I have known ten drops in this way cure an attack and give the patient a good night, simply by just putting her off to sleep. I do not think that any amount of asthmatic dyspnœa is any reason against giving it, or constitutes in any degree an element of danger—supposing, that is, that the Asthma is pure, and that the dyspnœa is neither cardiac nor bronchitic. In either of these cases the dyspnœa, being organic and not of a nature which the chloroform would remove, would constitute a serious embarrassment; whereas the asthmatic dyspnœa would cease to exist, and therefore cease to be any source of danger, in just such proportion as the influence of the chloroform was established.

Nitre Paper.—This is perhaps now one of the best-known and best-established remedies of Asthma, as it is one of the most uniformly successful. So generally is it efficacious, that it is always a matter of surprise to me when an asthmatic tells me that it does him no good. I am not certain of the category in which I ought to place it, and I class it among sedatives, and am inclined to think that it acts as one, chiefly on account of the strong soporific influence that it exercises. It affects not only the patient in this way, but the bystanders. On the very day that I am writing this, a lady has complained to me that she finds it almost impossible to administer the nitre fumes to her husband, on account of the irresistible sleepiness with which it overwhelms her; and I have mentioned in my work on Asthma the case of a lady who burnt the paper every night of her life in bed, but always had to wake her husband up, as soon as the fumes had relieved her breathing, because they made her so helplessly drowsy that she feared she might fall back while the paper was still burning, and set the bed on fire; she always *did* fall back asleep before the process was over, and her husband always had to take charge of the embers. What are the exact products of the burning of nitre paper I do not know, nor of those products what may be the remedial one, or ones. This is a subject that still waits investigation. The papers may be made by the patient (by dipping ordinary blotting-paper, white or red, into a warm saturate solution of saltpetre), or bought at any chemist's. The papers should be kept in a dry place, so as always to be fit for use. When employed, a piece about six or eight inches square should be torn off and lit at one corner. As the ignition fizzes along the edge of the paper, white fumes arise which are to be inhaled. I do not think it necessary or advantageous that the actual smoke itself should be drawn into the chest, but the air in its immediate neighbourhood which is impregnated with it. It is a good plan to burn the paper in a small room, or confined space, so as to get the air thoroughly charged with the fumes; a cupboard, or closet, or four-post bed, with the curtains close drawn, answers very well; I have seen a patient

make use of a large carriage umbrella for this purpose. There are two ways in which the paper may be advantageously used:—one habitually at stated periods, as a preventive, as, for example, every night and morning; and the other when the Asthma shows itself, with a view to its immediate relief. By using it in the former way, patients may often prevent the development of any attack for a long period. For example, many persons burn the paper every night in their bedroom on going to bed, and retire to rest with confidence and with the certainty of immunity through the night; whereas, if they go to sleep without first impregnating the air of their bedrooms with the nitre fumes, they are as certain to be disturbed with their Asthma. Others, with a view to its use in the latter way, always carry some nitre papers about with them wherever they go, and if their asthmatic symptoms appear burn a piece, and in a few minutes are relieved. Such patients never go to bed at night without having some of the paper by their bedside, that, if their Asthma disturbs them at night, they may immediately resort to their remedy. So rapid are its effects often in these cases, so complete is the relief, and so drowsy do the combined effects of the previous dyspnœa and the nitre paper render the patients, that they have not time, as I have already mentioned, to put the still burning paper in a place of safety before they fall back asleep.

Stimulants.—This is a class of remedies whose action is very different from, one may almost say opposite to, the action of those I have just mentioned, but which nevertheless exercise a most powerful influence over the asthmatic state. Among these, *coffee* is perhaps the best known, and the most generally efficacious. I find, in the majority of cases of Asthma that come before me, that coffee has been tried, and that it has given relief. It should be made as strong as possible, *café noir*, taken as hot as it can be swallowed, without either milk or sugar. It should also always be taken upon an empty stomach; coffee taken with food not only does no good, but does positive harm, by impeding the process of digestion. I have known more than one case, as I have mentioned elsewhere, in which coffee made in the ordinary way, and taken immediately after dinner, had a strong tendency to induce Asthma, although, taken in the way I have above described, it had a very powerful beneficial influence.

Alcohol, in its various forms, is another remedy of this class, that my experience during the last few years has induced me to think highly of as a remedy for Asthma. In many cases it does not do much good, but in some it has a most powerful effect, and these I have noticed are frequently cases in which all other remedies have failed. In such cases I should certainly, if for this last reason alone, recommend its use; in any case where other remedies answered I do not think I should, on account of the many manifest objections there are to the habitual use of the stronger forms of alcohol. I have observed that it seems of little use unless given hot and strong—about half spirit and half boiling water; this circumstance seems to make more difference

than the kind of spirit, or the actual quantity taken. Some asthmatics prefer brandy, some whisky, some gin; but in all, however small the quantity of spirit taken, it must be hot and concentrated. The worst of this remedy is that it is so apt to become habitual, and to require to be given in larger and larger doses.

While speaking of stimulants, I think I ought to mention the curious and striking remedial effect that sudden emotion has in Asthma. There is nothing that suspends the asthmatic state so completely and so immediately. At once, without any gradual subsidence, the patient will pass from the most violent paroxysm to a state of perfectly free and unimpeded breathing. And this is the case not only in emotional temperaments, but in all kinds of people, of both sexes, and at all ages. The emotion may be pleasurable or painful, but it must be intense, and I think it acts more powerfully if it is sudden. Did the length of this paper permit, I might relate some very curious and interesting cases in illustration of this point, but I must content myself with merely mentioning the fact. And surely, if it were wanted, we could not have a more striking or convincing proof of the nervous nature of Asthma; I should myself want nothing more to establish this theory of the disease than this single therapeutical fact.

I have hitherto been speaking exclusively of the treatment of the paroxysms. But a very important part of the treatment of Asthma, indeed the only radical treatment of the disease, is the treatment in the intervals—that which is directed to the prevention of the attacks altogether. This is the only treatment that deserves the name of curative; the treatment of the paroxysms is but palliative.

There are, I think, three forms of treatment that have for their object this final cure of the disease by the prevention of the paroxysms. The first, the treatment by air—that is, by locality; the second, dietetic treatment and the regulation of the digestive organs; and the third, treatment by the avoidance of the excitants of Asthma, such as hay, animal emanations, &c. These different plans of treatment are applicable to different classes of cases; but if we examine them closely we shall see that they all really belong to one kind of treatment, that they all essentially consist in the avoidance of the provocatives of the attacks; their applicability depending upon what, in each particular case, is the special exciting cause.

Treatment by Air.—It has long been known to those who have either observed or experienced Asthma, that locality exercises a most remarkable control over the disease—that there are certain airs in which the asthmatic cannot breathe, and that there are certain other airs in which he enjoys a sure immunity from his malady; that, in fact, his being an asthmatic or not depends entirely on where he lives: if he lives in the one place, he is constantly suffering, but he might live twenty years in the other and never have an asthmatic sensation. There are some circumstances with regard to this curious fact that are constant, and worthy of note. In the first place, the effect is *immediate*; let the asthmatic be suffering ever so severely, he no sooner arrives at the air

that, in his case, is curative, than he is at once relieved. In the second place, the effect is invariable for each particular case ; there is nothing irregular or haphazard about it; the same thing may be repeated twenty times, and always with the same result ; so much is this the case that the asthmatic knows he may calculate on it with the greatest safety. I have mentioned elsewhere the case of a gentleman who, let him be suffering ever so much at Cambridge, would accept an invitation to a dinner party in London, knowing that as soon as he arrived in town he would be well. And this may go on for a lifetime, and is as noticeable in the production as in the cure of Asthma. A person may have an attack of Asthma on going to a particular place ; twenty years after he may revisit that place, and he will again be attacked. Again, the effect is *permanent ;* as long as the patient resides in the curative air, he is free from his disease, if it is for the rest of his lifetime, but only so long as he resides there ; for the remedy does not eradicate the asthmatic tendency; the patient has only to be exposed to the same influences as before to have all his old symptoms return upon him in their original force, and that after any lapse of time during which they have been suspended. Another noticeable point in most cases is the inscrutable character of the atmospheric peculiarity on which this influence depends, and very often its extreme slightness : the fact only is known that in such an air the Asthma never appears; but what is the peculiar character of that air, or in what respect it differs from another in which the patient cannot breathe, neither the asthmatic, nor his friends, nor his medical advisers, can even guess.

But while the effect of locality is constant for each particular case, the experience of one case is not the slightest guide for another ; on the contrary, there is the utmost diversity and contrariety with regard to this circumstance in different cases. The air that is a certain cure to one is death to another. One patient is best in the country, one in town ; one is best in an elevated position, one in a low one ; one is relieved by a relaxing air, one by a bracing ; one is best at the seaside, one inland. But though there is this uncertainty and irregularity, yet on the whole, on the average, there are certain rules as to what is curative. Thus, in the great majority of cases, an urban air is the air that cures, and of a city air that seems to be the best which is the most urban—the densest and smokiest. As a rule, the air of a low situation is better than that of a high one, and a relaxing air than one that is bracing. In some cases there is one place, and only one, where the Asthma manifests itself. In such cases the circumstance has generally been discovered by accident—the asthmatic has suddenly been seized, soon after his arrival at some place that he never visited before, with strange and alarming symptoms which have turned out to be Asthma. These symptoms may never again appear, except on a return to the same locality. It is, however, much commoner for there to be many places where the Asthma is apt to occur, and only one, or but few, in which the asthmatic tendency seems to be in abeyance.

Treatment by food is the sovereign and final treatment of all those cases in which the Asthma is produced, and only produced, through the stomach. There are many cases in which a late dinner, or a supper, is sure to bring on an attack, but in which nothing else will. In such cases the patient has only to abstain from food after an early dinner, and he will see no more of his Asthma for such time as he keeps up such abstention, if it is for the term of his natural life. Such a person may cease to be an asthmatic at pleasure —that is, he ceases to be an asthmatic *in esse*, not *in posse;* for immunity so obtained does not destroy the asthmatic tendency : let him at any time break through his rules, and his Asthma will immediately reappear.

Treatment by the avoidance of special provocatives is, as I have already mentioned, but the application to other cases of the same principle as the treatment of peptic cases by dietetic rules. Some patients always have Asthma brought on by hay, some by the smell of flowers, some by emanations from particular animals, such as cats, or dogs, or horses. Such persons have merely to keep themselves out of reach of the especial exciting causes, and they may elude their disease for any length of time. The radical treatment of bronchitic Asthma belongs to the same category, and consists essentially in the treatment of the bronchitis. Place such a patient under such circumstances as preclude the bronchitis, and with the cause you preclude the result ; send such a case to Australia, and there is an end cf his Asthma, because there is an end of his bronchitis.

PHTHISIS PULMONALIS.

By John Hughes Bennett, M.D., F.R.S.E.

Definition.—By the term Phthisis or Consumption (from φθίω to waste or consume) has been understood from the earliest times a disease characterised by wasting or emaciation of the body. The cultivation of morbid anatomy having determined that this condition was frequently dependent upon the deposition of little grains or nodules of a peculiar substance in the lungs, these received the name of tubercles. Thus the terms tubercle, tubercular disease, or tuberculosis, gradually came to be regarded as synonymous with Phthisis, which may now be said to comprehend all kinds of disease essentially connected with or dependent upon pulmonary tubercle.

It is this important morbid condition which we propose to describe in the present article, under the general heads of Pathology, Symptoms, Diagnosis, Prognosis, and Treatment.

I. Pathology of Tubercular Phthisis.

The pathology of Phthisis involves a consideration of the histology, chemistry, and general pathology of tubercle—of the morbid anatomy of the disease—of its causes—of its natural progress—and of the theory of its production.

Histology, Chemistry, and General Pathology of Tubercle.— The term tubercle literally implies a little swelling, and in this sense it still serves to distinguish a class of skin diseases. As applied to the peculiar deposits so frequently found in the lungs and other organs, it now means not only those products when they present a tubercular form, but when they are infiltrated in masses, or exhibit appearances wholly opposed to the original signification of the word. At present, by tubercle is understood a peculiar morbid deposit, sometimes grey, but more frequently of a yellowish colour, varying in size, form, and consistence, which sometimes softens, and causes ulceration in the surrounding textures, but which at others dries up, becomes cretaceous or calcareous, and produces induration and cicatrization.

The ultimate structure of tubercle varies according as it is soft or hard, or as it has been recently or for a long time deposited. If we mix a minute fragment of yellow, tolerably soft or cheesy, tubercle

with a drop of water, and crush it between glasses, so that it may be thoroughly broken up, and capable of being examined with a magnifying power of 250 diameters linear, it may be seen to consist of a number of irregularly-shaped bodies, and of numerous molecules and granules. The bodies are called *tubercle corpuscles*, and approach a round, oval, or triangular form. Their longest diameter varies from the four-thousandth to the two-thousandth of an inch. They are solid, having a distinct external outline, and have embedded in them generally three or more granules and molecules, varying in size from a point scarcely measurable to the six-thousandth of an inch in diameter. Acetic acid causes partial solution and transparency of these bodies. Æther and alcohol produce little change. Ammonia and Liquor potassæ cause them to break down and dissolve with varying rapidity. The molecules and granules differ greatly in various specimens of tubercle, sometimes being very minute, and at others half the size of the corpuscles themselves. Chemically, they may be albuminous and partially soluble in acetic acid—fatty when they are soluble in æther and potash—or mineral when they are dissolved by the mineral acids.

The corpuscular and molecular elements of tubercle are always present, but in different proportions. Generally speaking, in indurated or grey tubercle there are few molecules, and the corpuscles are so compressed together as to be scarcely distinguishable. On the other hand, in soft tubercle the molecules are numerous, and the corpuscles easily separable. The more tubercle softens and becomes diffluent, the more the relative amount of the molecular element increases.

In chronic tubercle, and especially when it has undergone the cretaceous or calcareous transformation, the elements described become mixed with hard, gritty particles of earthy salts. These are of irregular form and size, and are large and numerous in proportion as the tubercle is more and more calcareous. They are often associated with crystals of cholesterine, and not unfrequently with black pigment granules and masses. When tubercle is converted into a mass of stony hardness, a thin section of it presents an irregular granular appearance, made up of a congeries of minute earthy particles without any distinct form.

Tubercle corpuscles may be associated with pus and granule cells, as well as those peculiar to glandular organs or mucous surfaces. From pus corpuscles they are readily distinguished by the action of acetic acid, which in them causes no granular nucleus to appear. From the fibre or plastic cells found in recent lymph they may be separated by their irregular form, smaller size, and the absence of primitive filaments. With the granule cell they can scarcely ever be confounded on account of its large size, brownish appearance, and granular structure. From gland or epithelial cells they are distinguished by their smaller size and the absence of nuclei. Cancer cells also are at once recognised by their size, transparency, and oval nuclei. The only elementary structures resembling tubercle corpuscles are those constituting the

reticulum of cancer and the disintegration of fibro-nucleated growths. The former, although often, even to the naked eye, resembling tubercle, and under the microscope composed of irregularly-shaped nuclei, and numerous molecules, resulting from the histolysis of cancer, are almost always associated with the more recent cell-forms of that growth, while the fragments or presence of fibres serve to distinguish the latter. It should be remembered that all forms of exudation, and many kinds of growth, at an early period of development, present a molecular and nuclear structure throughout, and might by inexperienced histologists be confounded with tubercle. A careful consideration of all the circumstances connected with tubercle, and of the distinctive structures associated with it, however, will seldom deceive the skilful observer.

Tubercle has been made the subject of special chemical analysis by numerous chemists, from which the following conclusions may be drawn :—1. That it consists of an animal matter, mixed with certain earthy salts. 2. That the relative proportion of these varies in different specimens of tubercle. That animal matter is most abundant in recent, and earthy salts in chronic tubercle. 3. That the animal matter consists principally of albumen, occasionally mixed with a small amount of fibrin. Fat also exists to a slight degree, and becomes more abundant as a constituent as the disintegration of tubercle progresses. 4. The earthy salts are principally composed of the insoluble phosphate and carbonate of lime with a small proportion of the soluble salts of soda. 5. That very little difference in ultimate composition has yet been detected between tubercle and other albuminous compounds.

From the preceding structural and chemical facts tubercle must be regarded as a morbid product, having a very low degree of vital power, seldom proceeding beyond an imperfect degree of nuclear formation, and having a constant tendency to fatty or mineral degeneration. It assumes four forms :—

1. *Miliary Tubercle*, when the morbid deposit is scattered throughout an organ, or on the surface of a membrane, in isolated grains like millet seeds. Sometimes they are sprinkled indiscriminately throughout a tissue; at others, they are in groups or clusters more abundant in one part than in another. Occasionally they are minute, of greyish colour, semi-transparent, and hard to the feel—the so-called *grey granulations* of Bayle. More frequently they are of a yellow colour, about the size of a millet or mustard seed, and of soft consistence, so that they can be easily crushed between the fingers. In consistence they may vary greatly, being sometimes hard, or, as they are then called, crude, or they may be so soft as to resemble cheese and cream. They may have undergone the cretaceous or calcareous transformation, and still preserve their miliary form.

2. *Infiltrated Tubercle* occurs in diffused masses, varying in size from that of a bean to that of the entire organ affected. Thus a lymphatic gland, or the lobe of a lung, may present a uniform deposition of the substance throughout its whole extent. Between these two

extremes every variety in extent of deposition may be observed, masses being frequently formed by the agglomeration or condensation of miliary tubercle. Like it, also, this form of the deposit may be grey or yellow, crûde or soft, and undergo the cretaceous and calcareous transformation.

3. *Nodular and Encysted Tubercle.*—This form of tubercle exists in rounded, isolated masses, varying in size from that of a small pea to a bean. It may present all the characters of the other forms, but is frequently seen to be surrounded by a capsule, more or less dense, of fibrous tissue.

4. *Cretaceous and Calcareous Tubercle.*—This form of tubercle is distinguished by its white appearance, and its putty-like, gritty, or stony consistence.

All these forms of tubercle run into one another, and may exist in the same individual, and often in the same organ, especially in the lungs. They indicate no further essential difference in the nature of the deposits than is concerned with its amount and extent, its hardness or softness, its colour—whether white, yellow, grey, or black, or its being recent or old—miliary and infiltrated tubercle being generally new, while encysted and calcareous tubercles are always chronic. In the last the animal matter has been absorbed, while the mineral matter remains to form a concretion.

Great discussion has taken place as to whether tubercle is peculiar to any particular elementary tissue, and as to how it is produced. Like all forms of exudation, it may occur in every vascular texture, and readily coagulates in the minute spaces between or outside the textural elements immediately external to the vessels. Of this we may easily be satisfied by studying its special histology in various organs.

With regard to its mode of production, tubercular matter is first separated from the blood-vessels as a fluid exudation, forming by its coagulation a molecular blastema. The molecules of which it is composed then aggregate or melt into each other to produce the tubercular corpuscles. These, if compressed together and formed slowly, constitute the indurated dense granulations described by Bayle ; but if separated by soft molecular matter, produce the more common yellow miliary tubercles. The idea that these bodies are invariably the result of cell proliferation originates from the erroneous hypothesis maintained by Virchow and his followers, viz. that all morbid products are derived from cells. In their attempts to maintain this view, they have mistaken the occasional enlargement and proliferation of fibre cells in areolar tissue first described by Lebert, as fibro-plastic cells, for tubercular granules, which they describe as the essential elements of the lesion. It is not in the pleura or peritoneum, however, where such fibrous growths are occasionally seen, that the real manner in which tubercle is formed can be well observed, but in the lung, where the disease is most common and best characterised. There, all observation demonstrates that it originates in a molecular exudation, which, in consequence of diminished vital power, seldom passes beyond the

nuclear stage of growth. It is this low type of *hysto-genesis* that communicates to the exudation those essential characters which form the foundation of tubercular or phthisical disease.

MORBID ANATOMY OF PHTHISIS PULMONALIS.—Although tuberculization of the lungs is a constant and essential element of Phthisis, it rarely, if ever, happens that the disease proceeds to a fatal termination without affecting other organs. Nothing, also, is more common to find, during the examination of dead bodies generally, than that the lungs are often the seat of tubercle to a greater or less extent, although during life the presence of the disease has never been suspected. So common, indeed, is this lesion, and so many have been the able investigators of the alterations it produces in the various organs of the body, that all the anatomical facts connected with it may be said to be thoroughly known. We shall notice the morbid changes observed in cases of Phthisis in the different parts of the frame, *seriatim.*

The Lungs.—These are the organs in which, according to the researches of Louis, tubercle is sure to be discovered, if it occur in the body at all. This law, though now known to admit of some exceptions, especially as regards tubercular peritonitis, is still so generally true as to be one of the most valuable generalizations ever arrived at in pathological science. To the same distinguished physician we are indebted for another fact of no less importance, viz. that when tubercle occurs in the lungs it attacks the apices of those organs first. The exceptions to this law are so few as in no way to invalidate its great practical value.

The morbid changes found in the lungs of those who die labouring under Phthisis pulmonalis vary according as the disease is acute or chronic, as it is advancing or retrograding, and as it is associated with other lesions. In acute cases miliary and infiltrated tubercles are more or less general in one or both lungs. The deposit is generally soft, and frequently diffluent, causing ulcerations and irregular anfractuous cavities. The intervening pulmonary texture is often engorged with blood, is more or less pneumonic, while the bronchi are loaded with purulent matter. The acute disease in many respects resembles anatomically grey hepatization of the lung, and like it is more frequently most developed in the lower lobe.

In chronic Phthisis, constituting the vast majority of cases met with, all the forms of tubercle previously described are met with. The tubercle is most abundant at the apex, but may invade the greater portion of one or both lungs. In the latter case, it will most often be observed that one lung is more affected than the other, so that an examination of them displays all stages, either of the onward or retrograde progress of the disease; these, although often associated together in very chronic cases, are so distinctive anatomically as to require a separate description.

The appearances of the lung during the onward progress of the disease are—1. The presence of miliary tubercle to a greater or less extent. 2. The softening of this tubercle, so that it readily breaks

down under the finger or a current of water, and forms small cavities or irregular ulcerations communicating one with another. 3. The existence of distinct ulcers, excavations, or cavities, as they are named. These vary in size from a pea until they involve nearly the entire lung. There may be one or several. They may be isolated or anfractuous, that is, communicating with one another. If recent, the internal walls are irregular and rough; but if chronic, the ulcerative process has dissected out the fibrous tissue, leaving irregular bands stretched across the interior, composed of blood-vessels, the bronchi, or indurated fibrous tissue. When very chronic, the interior is lined with a smooth membrane. These cavities may be filled with air and fluids in varying proportions; the latter being viscous, purulent, occasionally sanguinolent, and not unfrequently ichorous, of a dirty-green colour and offensive odour. These changes in the lung may be associated in varying proportions with many other lesions to which the organ is subject. Pleuritic adhesions, by means of fibrous lymph, are very common; the pleuræ, at the apices of the lung, often being united to each other by a dense, tough substance which renders their separation impossible. Bronchitis, in all its forms and stages, may exist together with more or less emphysema, dilated bronchi, and collapse of the lung. There may be pneumonia or extravasation of blood, involving varying amounts of lung tissue.

There is a disease frequent in coal-miners, called carbonaceous lungs or Black Phthisis, in which there is no tubercle, but a deposition and infiltration of lamp-black or carbon in a finely molecular form, and which gives rise to cavities and disorganization of the pulmonary tissue, also commencing at the apex. It is accompanied by black spit, and is generally fatal.[1]

The retrograde progress of the disease is characterised anatomically, first, by the horny induration and cretaceous or calcareous transformation of the tubercular matter; secondly, by puckerings and cicatrices of the lung tissue; and thirdly, by contractions, loss of substance, and more or less induration of the organ. It may be observed in about one-fourth of all those who are examined after death in our public hospitals, that the apices of the lungs contain one or more masses, varying in size from a millet-seed to a coffee-bean, of cretaceous or calcareous matter. That these masses were originally tubercle cannot be doubted by those who have had any experience in post-mortem examinations, the more so as in various cases such tubercle, whether in the miliary, infiltrated, or nodular form, may not unfrequently be seen to present the various stages of induration and horny hardness, approaching towards the calcareous substance. Such hard masses if dug out and allowed to dry, indeed, become cretaceous, the animal matter having shrunk away, leaving the mineral substance unaltered. In old persons above seventy years of age, it has been shown by Rogér and Boudet that the presence of these concretions in the lungs

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[1] See the author's Clinical Lectures, 5th edit. "On Carbonaceous Lungs," p. 756.

increases to the extent of from one-half to four-fifths of all those examined.

If these concretions or masses of indurated tubercle occur at the surface of the lungs, the pleuræ covering them and subjacent tissue are frequently drawn in and puckered. If they occur deeper, they are surrounded by indurated pulmonary texture, more or less tinged of a black colour. Occasionally, also, linear and radiating cicatrices indicate the disappearance and closure of pre-existing ulcerations. Sometimes, however, tubercular cavities, instead of closing and forming cicatrices, remain permanently open and filled with air. They are lined by a smooth membrane, and almost always communicate with a bronchial tube. In this condition we discovered, in 1842, in such a case, associated with pneumo-thorax fungi growing in the infiltrated matter lining the chronic cavities, and have found them frequently in similar excavations since then.[1] At other times the bronchial tubes are permanently dilated, by the contraction and induration of the pulmonary tissue between them. This occurrence, conjoined with the other lesions referred to, gives rise to that condition described by Dr. Corrigan as cirrhosis of the lung.[2]

The various alterations now described may be associated with other lesions, especially chronic adhesions of the pleuræ, emphysema, chronic bronchitis, and dense pigmentary deposits. Not unfrequently it may be observed that whilst one portion of the same lung presents a marked example of the retrograde progress of Phthisis, another portion as decidedly shows the progressive changes. In such a case the former indicates tolerably well the older and more chronic transformations of the pulmonary tissue.

It would thus appear that there is nothing essentially destructive or necessarily fatal in Phthisis, and that in all stages of the disease it may be checked, and enable the individual affected to live many years subsequently, and die of old age or other disorders. Attention to morbid anatomy in recent times is demonstrating that this occurs far more frequently than was formerly supposed, and is due not only in many cases to the spontaneous efforts of nature, but in not a few to the direct interference of art.[3] This latter termination, however, is materially interfered with should other organs participate in the disease; and the morbid changes observed in them, therefore, next demand our attention.

The Pleuræ.—We have already pointed out that during the whole progress of Phthisis the pleuræ, as well as every other part of the lung, are apt to be affected. This, however, may not only be exhibited by adhesions more or less dense, but not unfrequently by the deposition of tubercle

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[1] Description of a Cryptogamic Plant found growing in the sputa and lungs of a man who laboured under pneumo-thorax. Trans. of Royal Soc. of Edinburgh, 1842.

[2] See Dublin Medical Journal, vol. xiii. 1838; Laennec, vol. i. p. 201; Reynaud, Mémoires de l'Académie, tome 4ᵐᵉ; Cruveilhier, Anatomie Pathologique, livraison 32, planché 5, fig. 3; and the author on Pulmonary Consumption, 2d edit. Case 3, p. 57.

[3] See the author's work on Pulmonary Consumption, in which several such cases are recorded, and the post-mortem appearances figured,—Figs. 21 to 26.

in a miliary or infiltrated form, the latter of which assumes a laminar or stratiform character. Effusions and exudations into the pleural cavity may also occur, giving rise to more or less hydro-thorax and empyema. Further, the pulmonary pleuræ may be ulcerated and communications take place with tubercular cavities, or with the bronchial tubes, in which last case pneumo-thorax is the result.

The Trachea and Larynx.—The trachea and larynx are very commonly the seat of congestion and ulceration in cases of Phthisis. In the mucous membrane of the former the ulcerations are frequently small, numerous, and round, as if dug out with a small point; at others, they are larger, deeper, and lay bare the cartilaginous rings. In the larynx they are generally irregular, varying greatly in size, and sometimes involving both vocal cords and the whole interior of the organ. Their edges are occasionally studded with indurated tubercles, and sometimes there is thickening with œdema of the cellular tissue, tending to close the glottis. In chronic cases of laryngeal ulceration, which is often called *Phthisis laryngea*, caries and necrosis of the cartilages may occur.

The Bronchial Glands.—It is seldom in cases of chronic Phthisis that the bronchial glands escape being affected with tubercle, which assumes the infiltrated form, and causes in these considerable enlargement. On section they may be almost colourless, but they are sometimes more or less loaded with black pigment.

Heart and Pericardium.—It is very rarely that tubercle is deposited in the heart or pericardium, but when this does occur, it takes place in the nodular form. The heart, however, is very liable to become atrophied, and smaller than usual. In lingering cases of the disease, with extreme emaciation, it may be found after death not larger than a duck's egg. The bulk appears to be adapted to the diminished amount of blood in the body, and the little work it has to do.

Alimentary Canal.—Very rarely ulcerations may exist in the pharynx, but enlargement of the follicles is common. The œsophagus and stomach are organs which are remarkably free from tubercular disease; but, according to Louis, the mucous membrane of the latter viscus is liable to softening, mammilation, and attenuation, in the majority of cases. In the small intestine the glands of Peyer are very liable to enlargements and ulceration, especially in its lower third. The enlargements are owing to the deposition of tubercle in and around the solitary and aggregated glands, often accompanied by considerable redness and vascular congestion. It presents the miliary or granular forms, although occasionally it may exhibit small nodules the size of peas. Tubercular ulcerations of the small intestines are common in the last stages of Phthisis, and occupy the seat of the solitary and aggregated glands. In the first case they are rounded, with abrupt or tuberculated margins, with a yellowish or dirty-grey base. In the latter case they are oval in form, running transversely round the gut, so that they are readily distinguished from typhoid ulcerations, the long axis of which is in the opposite direction. Their

margins are smooth, sometimes tuberculated; the base sunk, and covered with a purulent or dirty-greyish substance. Above the ileo-colic valve the ulcers have a tendency to run into one another and produce an ulcerated surface, more or less broad, surrounding the gut. These ulcers occasionally are so deep as to perforate the intestine. Similar tubercular granulations and ulcers may also be found in the large intestine.

Peritoneum.—In rare cases the intestinal ulcerations perforate the bowel, almost always giving rise to fatal peritonitis. Not unfrequently, however, chronic adhesions exist, to a greater or less extent, on the peritoneal surface outside these ulcers, uniting coils of intestines to one another, or to the abdominal walls. Chronic tubercular peritonitis may also occur when the intestines are covered and agglutinated together by coagulated exudation, studded throughout with miliary tubercle. This lesion, though it may accompany Pulmonary Phthisis, may, sometimes, exist as a primary and independent disease.

Mesenteric and other lymphatic glands.—These are very liable to be enlarged in Phthisis, when they may present greater or less induration and enlargement, according to the recent or chronic condition of the disease. Tubercle usually is present in them in the infiltrated form, at first yellow, cheesy or soft, afterwards white and indurated, and, in a few cases, cretaceous and calcareous.

Liver.—In children the liver is not unfrequently the seat of miliary and infiltrated tubercles, but in the adult this is very rarely observed. More commonly the organ is enlarged, a result previously supposed to be owing to fatty degeneration, but now known to depend upon a peculiar albuminous transformation known as the waxy disease, from its resemblance to bees'-wax. In this condition it may be so enlarged as to weigh eight or ten pounds. It presents a peculiar density to the feel, a pale fawn or yellow-brownish colour; and on section the cut edges, when held up to the light, are semi-translucent. We were the first to examine this disease of the liver microscopically in 1845, and found the hepatic cells to be condensed together, shrivelled, colourless, and of peculiar transparency, with the nucleus absent, or evidently disappearing.[1] It has been supposed by some to be related chemically to starch, and therefore called amyloid degeneration. But it is never changed blue on the addition of iodine, although we have found that like certain other forms of albuminous compounds it possesses the property of fixing colours, such as the reddish-brown tint of iodine, or the peculiar pigments of indigo and carmine.

Spleen and Kidneys.—Both these organs, like the liver, in early life may become subject to tubercular deposits in the miliary form, which in the adult are very rare. The kidney further is liable to extensive tubercular deposits, causing abscesses, or what is known as scrofulous pyelitis. Like the liver also, it is commonly affected in Phthisis with the waxy degeneration, causing induration and enlargement of

[1] See the author's Clinical Lectures, 5th edit. Case clxi. p. 731. Also remarks on the waxy degeneration, Idem, p. 249.

its substance, and the same translucent albuminoid degeneration of the cells and vascular elements.

Other textures and organs.—In the foregoing summary we have only shortly alluded to the morbid changes most commonly found in cases of Phthisis. It should be understood, however, that almost every vascular tissue in the body may, under particular conditions, be subject to tubercular deposits in conjunction with the disease of which we are treating, and thus, in special cases, the bones, muscles, the brain and its membranes, skin, the bladder, testes, &c. &c., may be occasionally involved.

CAUSES OF PHTHISIS PULMONALIS.—The various circumstances which predispose to Phthisis have been most anxiously investigated. All we can venture to offer in this place is a very general summary of the numerous researches undertaken in connexion with this subject.

Age.—Phthisis is not a disease that is common in early infancy or in advanced age. It is more frequent during childhood and youth, although cases may be seen in many persons of middle age, as well as among young children. From the returns of the Brompton Hospital for Consumption, it would appear to be most frequent between the ages of twenty and thirty. Age unquestionably greatly influences the progress of Phthisis, the acute being most common in young, and chronic in elderly persons. We should not forget, however, that Phthisis in advanced life is frequently the termination of a prolonged case, which commenced many years previously.

Sex.—It is generally supposed that Phthisis is more common in females than in males, but this does not appear to be an invariable rule. It is certainly not the case in the Royal Infirmary of Edinburgh, Dr. Home having pointed out that in the years 1833, '34, and '35, 185 cases were males, and only 112 females. The same excess of males labouring under the disease has prevailed in that institution ever since, as in the years 1843 to 1846 inclusive there were—males 356, females 217; and in the latest reports for the year 1865 the numbers are—males 126, females 64.

Hereditary tendency.—Instances are not uncommon in which members of the same family are observed to become affected one after another with Phthisis, on arriving at a certain age. This, however, may depend not so much upon weakness inherited from parents, as it does upon a vicious method of rearing the infants and children of certain families. We have seen the children of many families become phthisical, in whom no hereditary taint could be traced, and have frequently pointed out, in the clinical wards of the Royal Infirmary, that, among the six or eight cases of Phthisis then present, not one could be traced to hereditary causes. Although, therefore, there can be no doubt that weakness in parents is a cause of weakness in the offspring, we are of opinion it is by no means so general or influential a source of Phthisis as is usually supposed.

Vitiated atmosphere.—This has been concluded to be a powerful

cause of Phthisis by numerous authors, and there can be no doubt that the habitual breathing of deoxidized or impure air must greatly impede nutrition. Among the poor there can be little difficulty in attributing its effects to close or overcrowded rooms, in which they work and sleep. Among the higher classes this is not so obvious a cause, although Baudelocque, in support of this his favourite theory of the origin of tubercles, accused them of lying in bed too long, and said that the children slept with their heads under the bed-clothes.

Climate.—It is an undoubted fact that Phthisis is more frequent in temperate climates than in very cold or very warm ones. It is by no means common in Russia and Canada, notwithstanding the long continued cold, nor does it prevail among the nations of the tropics. These last, on the other hand, are peculiarly liable to Phthisis on coming to Europe. Some favoured spots are stated to be free from Phthisis; among these, it has been recently pointed out by Drs. Macrae and M'Coll, are the islands of Lewis and Mull, among the western isles of Scotland. Dr. Hjaltelin has informed me that Iceland enjoys a like immunity.

Contagion and Infection.—Several of the older writers were of opinion that Phthisis was contagious and infectious, an opinion still widely disseminated in certain countries, more especially Spain and Italy. We have too frequently seen the death of a phthisical patient in Italian hotels give rise to the most extortionate demands for the pretended destruction of bedding and furniture, all of which should be firmly resisted. It has occasionally been observed that Phthisis in a wife or husband has been followed by the appearance of the disease in the husband or wife. The frequency also with which young women become phthisical after pregnancy has given rise to the idea that they may have been infected by the opposite sex through the uterus. These ideas have received no support from the profession. In 1865, however, it was announced by M. Villemin[1] that the cause of tubercle was a virus, and that he had succeeded in inoculating it in healthy rabbits, by inserting grey granular tubercle below incisions in their skins. These experiments appear to have been carefully performed. They have been successfully repeated by Lebert, and also by others with varying results. The experiments of Drs. Andrew Clark,[2] Wilson Fox,[3] and Burdon Sanderson[4] have further shown that not only tubercle but a variety of other morbid products, and even local irritation of the tissues, may produce deposits in the glands, lungs, and various organs in rabbits, and especially in guinea-pigs. Thus the introduction of a seton produced them in one case, and pieces of putrid muscle in no less than four out of five inoculations.[5] These facts show that the lesions described as tubercle are analogous to the secondary deposits

[1] For a good summary of M. Villemin's views and experiments, see Edinburgh Medical Journal for February 1867, p. 756.
[2] Medical Times and Gazette, 1867.
[3] On the Artificial Production of Tubercle in the Lower Animals. 4to. London, 1868.
[4] Tenth Report of the Medical Officer of the Privy Council. London, 1868.
[5] Wilson Fox, op. cit. p. 5.

occurring in pyæmia, and which are known to result from the poisoning of the blood by absorption and injection into it of putrid fluids, but they in no way support the hypothesis that Phthisis Pulmonalis is contagious or infectious. But we shall again allude to this matter under the head of Theory of the Production of Phthisis.

Occupation.—Phthisis is unusually common among the workers in certain trades, more especially stone masons, grinders and polishers of steel, dressers of flax and feathers, cotton carders, china scourers and potters, tailors, sempstresses, straw plaiters, lace makers, silk workers, and iron and coal miners. On the other hand, cooks, butchers, tanners, tallow chandlers, and soap boilers, enjoy to a great degree an immunity from the disease. In the first class of cases the inhalation of foreign particles into the lungs excites local irritation, which proves injurious to the respiration, and deteriorates the constitution; or the result is occasioned by the combined operations of sedentary employments, impure atmosphere, exhaustive work, and bad food. In the second class of cases there are good wages, and, as a concomitant, good food, while a constant contact with oil is supposed to offer an additional explanation of the fact.

Humidity has been supposed to exercise a considerable influence in the production of Phthisis. Magendie thought he had produced tubercle in rabbits by confining them in damp cellars. Baudelocque points to numerous localities, such as morasses, houses surrounded by ditches, and so on, where the disease is rife. It is also common in Holland, and other countries liable to damp fogs, and an atmosphere saturated with moisture. Phthisis has been shown to prevail in the damp soils of the United States by the careful investigations of Dr. Bowditch of Boston, U.S., and of England, by those of Dr. Buchanan.[1] On the other hand, in elevated dry regions it is said to be comparatively rare. In the Seventh Annual Report of the Registrar-General for Scotland, it is pointed out that for every 100,000 inhabitants there died annually from consumption 206 persons in Leith, 298 in Edinburgh, 310 in Perth, 332 in Aberdeen, 340 in Dundee, 383 in Paisley, 399 in Glasgow, and 400 in Greenock. In these towns, therefore, the death-rate is diminished in proportion to the dryness of the site.

Diet.—Of all the causes producing Phthisis and tubercular diseases generally, a low diet, or imperfect assimilation of food, is the most obvious and unequivocal. Among the lower orders we observe this to be the case in all large cities, among the ill-fed and half-starved poor, in orphan and foundling institutions, and whenever from any cause the food of the people is rendered scarce or dear. In the higher classes we observe it following the system of nourishing infants by hired nurses, or bringing them up by hand, and in early childhood from a pampered indulgence in indigestible or non-nutritious substances. Not unfrequently it results from allowing weak children to reject the fatty con-

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[1] Tenth Report of the Medical Officer of the Privy Council, 1868.

stituents of food. Most of the other causes to which we have referred will be found on examination to have influenced the economy, by diminishing appetite, and impeding digestion and assimilation of food.

Other diseases.—It has frequently been observed that Phthisis follows attacks of previous diseases, which by either affecting the lungs, or strongly depressing the system, and not unfrequently by both, appear to have caused the disease. Thus it has followed pneumonia, bronchitis, measles, and hooping-cough in persons previously healthy. Want of appetite and dyspepsia in the young are fertile sources of Phthisis. Indeed, all disorders which permanently lower the strength in the young, and interfere with the nutrition so necessary at that period of life for developing the growth of the body, may be regarded as a cause of tubercle. The weakness resulting from parturition and prolonged lactation in feeble women is a striking example. For the same reason it occurs in some rheumatic and gouty persons.

Predisposition.—Seeing that none of the causes mentioned invariably produce the disease, and that striking exceptions may be cited of persons who exposed to one or all of them have yet escaped the malady, the difficulty has been attempted to be got rid of by recourse to predisposition. In the same manner that many persons exposed to fever or small-pox are not affected, or that certain plants only grow on particular soils or patches of ground, so it is said there must be a something superadded to other causes in tubercular cases, which is called predisposition. It is unnecessary to enter upon the subtle argument which has thus been raised, and which appears to us, in the present state of science, as reasonable as is the calculation of chances concerning the probability of escape to any particular soldier who exposes himself to the fire of an enemy. In neither case is it predisposition nor chance, but rather the operation of fixed laws, which it is not given to us as yet to recognise, or regarding which we cannot so calculate as to avoid their operation.

It may be observed, especially among the lower classes, that vitiated air, humidity, want of cleanliness, bad diet, drunken habits, and a variety of debilitating causes, all concur apparently to produce the effects, so that it becomes very difficult to attribute the disease to any one especially. In the higher classes two causes more especially are found, viz. an hereditary taint, and improper nutrition. On looking at the whole train of causation, it seems to me certain that they may all converge in mal-assimilation or deficiency of food. As far as the strength of the economy and constitution of the blood are concerned, it matters little whether deficient vitality be caused by the food being deficient, or, if abundant, its not being digested, or again, if digested, it being deteriorated in the lungs by noxious gases, by inoculation of morbid matters, or by constant congestion, the result of tissue irritation. As a general conclusion we hold to the belief that the great cause of tubercle is weakness of constitution, or diminished vital power, however produced; a theory which has the merit of teaching mankind

to avoid all causes which may exhaust the frame, and to establish as remedies everything that can communicate to it strength and vigour.

NATURAL PROGRESS OF PHTHISIS.—The commencement of Phthisis may be said to be established as soon as it is distinctly shown that tubercles exist in the lung. This period, however, is generally preceded by more or less deterioration in the general health, indications of debility, and impoverishment of nutrition. It is true there are many individuals in whom the deteriorating process is so gradual, that this change has not been observed either by themselves or their friends, but it is seldom that such will escape the observation of the experienced physician. At other times the impaired health is caused by some exhausting malady of a general character, or of one especially affecting the chest. It sometimes happens that the first obvious departure from health is a hæmorrhage coming from the lungs. It is under these or other exhausting circumstances that a matter is exuded in a fluid state from the capillaries of the lungs, which collects and coagulates in such portions of the pulmonary texture as offer least resistance. Although a small portion may insinuate itself between the elementary textures of the organ, it will principally pass into the air-vessels, so as to obstruct the entrance of air. A miliary tubercle may in this way block up from three to twenty of these air-vesicles. The amount of isolated tubercles so formed in the lung, their aggregation and union together giving to the morbid product the appearance of infiltration, somewhat impedes respiration and the functions of the pulmonary organs, according to the extent of the morbid product. Their presence, also, by irritating the pulmonary nerves, gives rise to the frequent dry cough so common in the early stage of the disease. The tubercular matter having coagulated, constitutes a foreign solid body, which can only be removed by being again broken down and so rendered capable of being either absorbed or excreted. Thus the miliary or infiltrated forms, whether grey or yellow, after a time soften—a process which may commence at any part of the mass, and gradually affect the whole. This softening is a disintegration or slow death of the tubercular exudation, constituting true ulceration, which is more or less extensive, according to the amount of the morbid deposit. When recent, the pulmonary tissue in the immediate neighbourhood is more or less congested, but when chronic it is thickened and indurated, often forming a capsule, which surrounds the hardened tubercle, or a membrane lining an excavation. The other neighbouring tissues are also necessarily involved. The pleuræ are thickened, the bronchi sometimes loaded with tubercle, at others obliterated by pressure, the blood-vessels are congested, ruptured, and ultimately impervious, and the nerves compressed and irritated. As the ulcerative process extends, the elementary structures of the lung are more and more destroyed, the excavations become larger, more numerous, and unite with each other, until at length the pulmonary organs can no longer

perform their functions. In most cases, however, before this is arrived at, tubercle appears in other parts of the body, producing complications, under the united effects of which the strength is exhausted.

It is only in rapid or acute cases of Phthisis that the ulcerative tendency of the tubercular exudations pursues an uniformly destructive progress. In chronic cases this is frequently checked, and for a time slumbers, the symptoms improving and the patient exhibiting temporary signs of recovery. These arrestments of the disease may be of greater or less duration; and there can be no doubt that they are permanent in a far greater number of persons than is generally supposed. Indeed, while the more extended cultivation of morbid anatomy in recent times has demonstrated the frequency of cretaceous and calcareous concretions at the apices of the lungs, as well as of pulmonary cicatrices, physical diagnosis and more careful observation have shown in the living, that corresponding with the disappearance of symptoms and physical signs the health has improved, and ultimately been permanently restored. We are satisfied that there is no period in the history of the disease in which permanent arrestment may not take place, although, of course, it is far more common when it is limited in extent, and confined to one lung. The facts we have seen and recorded on this subject, however, show [1] that individuals with extensive cavities and disease on both sides may, under favourable circumstances and with appropriate management, ultimately recover.

THEORY OF THE PRODUCTION OF PHTHISIS.—It is not our intention to enter into an account, descriptive and critical, of the numerous views which have been held in past times as to the essential nature of Phthisis. It will be sufficient to speak of the two theories which are now being discussed, and of the reasons which induce us to adopt the one and to reject the other. The first theory supposes an altered condition of the blood, originating in a perversion of nutrition. This perversion, as we have seen, has been considered by some to be owing to vitiated air, by others to imperfect assimilation of food, and by others to an hereditary taint. It has also been shown experimentally, that it may be caused in the lower animals by inoculation of various morbid matters. All these, and indeed other causes, may originate or co-operate in diminishing the vital power of the individual, and directly or indirectly produce weakness, feeble digestion, and an impoverished blood. It is when in this condition that any accidental irritation of the lungs, often inappreciable and undetectable, causes a limited congestion here and there in the pulmonary organs, which terminates in more or less exudation of the liquor sanguinis. This exudation coagulating causes the miliary and infiltrated forms of tubercle previously described, which partaking of the diminished vital power of the organism, instead of being transformed into the pus characteristic of a similar exudation in a healthy person, produces the small, irregular, and imperfect bodies called tubercle corpuscles.

[1] See my work on Pulmonary Consumption, Cases 1, 2, 21, 22, &c.

Instead of cells, which are rapidly produced, broken down, and absorbed as in pneumonia, we have numerous molecules and bodies resembling ill-formed nuclei. In short, we have a chronic exudation, in which the vitality is so lowered that it tends to disintegration and to produce the lowest kind of organic forms,—*i.e.* molecules, granules, and nuclei.

The second theory is one which, instead of ascribing tubercle to an exudation from the blood, of low vital power, regards it as the result of increased cell development and multiplication of the included nuclei. According to this view tubercular matter is a new growth, which when we consider that it sometimes reaches the size of an apple, as in the brain, would demand for its production increased rather than diminished nutrition. Notwithstanding the desire of those who support an exclusive cell theory to trace tubercle as well as every morbid product to some cell transformation, the most careful and repeated investigations of histologists have failed to do so. According to Virchow, however, upon isolating the constituents of a tubercular mass "either very small cells provided with one nucleus are obtained, and these are often so small that the membrane closely invests the nucleus, or larger cells with a manifold division of the nuclei, so that from twelve to twenty-four or thirty are contained in one cell ; in which case, however, the nuclei are always small and have a homogeneous and somewhat shining appearance."[1] This description of small nuclei in the interior of cells, and the appearances figured as constituting the structure of tubercle, have, so far as we are aware, never been confirmed by any experienced histologist. Tubercle is so common a morbid product that if such indeed were its constitution, it ought to be seen at once ; but our most anxious and repeated efforts have failed to discover it, nor does there exist a single preparation anywhere capable o ꞏdemonstrating it. Cells containing many nuclei are very rare, associated with tubercle, and when they do occur are evidently dependent on the occasional irritation of texture which is produced around the morbid products—they are a result and not a cause. As a matter of fact, therefore, not to speak of the theoretical improbability of a disease originating in weakness commencing with increased power of vital development in the pre-existing tissues of the organism, this theory must be rejected.

In support of this last theory it is further maintained by Virchow and his followers, that the term tubercle should be limited to the minute, indurated granulations which, as Lebert originally pointed out, are the result of increased nuclear growth in the fibrous tissues— what he denominated fibro-plastic corpuscles. The larger so-called tubercular infiltrations of morbid anatomists and practical physicians they regard as chronic or, as they call them, cheesy exudations. Dr. Burdon Sanderson proposes that tubercle should be called an "adenoid growth,"[2] and it may be granted that a mass of molecules and tubercle

[1] Virchow, by Chance, p. 476, and fig. 140.
[2] See Edinburgh Medical Journal, November 1869, p. 386 ; and Eleventh Report of the Medical Officer of the Privy Council, plate 5, fig. 3.

corpuscles, such as we have described, in a fibrous tissue, may present a vague resemblance to one of Peyer's glands. But a slight consideration must show that these distinctions are more verbal than real. It is not the occasional, scattered, and rare indurated granulation with which we are so much concerned as the extensive, chronic morbid deposit. Transferring or limiting the term tubercle to the accidental granule, and calling the general and essential morbid product chronic inflammation, or adenoid growth, constitutes no real advance in pathology. What we have from the first maintained is that we have to do with a *tubercular exudation,* which differs from an inflammatory and cancerous exudation in its low vital energy, and diminished power of transformation into cell forms; and that this is the essential element of Phthisis Pulmonalis. Two recent French admirers of Virchow's doctrines have proposed to separate ordinary Phthisis from granular tubercle of the lungs, under the name of Tubercular Pneumonia,[1] and Niemeyer suggests for the term Phthisis chronic Pneumonia.[2] These propositions, while they indicate an essential agreement with the doctrines contended for in this article, offer no real advantage. It is not the name we attach to a morbid state, but a clear comprehension of the morbid state itself, which is of real importance. It is now many years ago that we pointed out the existence of a true vesicular pneumonia, which to the naked eye resembled scattered grains of yellow tubercle, but which under the microscope was composed of desquamated epithelial scales and pus cells, mingled with fine molecular matter.[3] That a pneumonia may be vesicular, lobular, or lobar, is now agreed upon by every pathologist, and the same forms dependent on the extent and seat of the exudation may be observed in tubercular deposits.

Satisfied then that tubercle is essentially a coagulated exudation, we have next to ask, why such exudation is not rapidly transformed into pus cells, as occurs in an acute pneumonia? The reply is, in consequence of the deficient strength and want of vital formative power in the organism. If it be further asked, on what that deficient energy, in its turn, is dependent? the answer is, that in consequence of impeded nutrition, or other causes, the blood is rendered so abnormal, that its fluid constituents when exuded are incapable of supporting cell formation. But it must not be forgotten that as the blood is continually undergoing changes, now receiving and then giving off new matters, it never remains the same for many hours together. An exudation at one period may abound in elements which do not exist in it at another. Hence why we find all kinds of intermediate formations in the textures in tubercular cases, and why the exuded matters associated with the lowest form of morbid formation may be occasionally mingled with the higher. A cancerous growth, however, is very rarely met with in conjunction with tubercle.

[1] Bérard et Cornil sur la Phthisie, 1867.
[2] On Pulmonary Consumption; Sydenham Society's Translation.
[3] Clinical Lectures, 5th edit. p. 689.

When we next come to inquire what is the nature and essential cause of that altered nutrition which so modifies the blood, that when its fluid portion is exuded it should constitute tubercle, we must inquire in what manner the digestive processes are primarily impaired. And here we must remember that all food essentially consists of albuminous, fatty, and mineral constituents, which are reduced in the alimentary canal to a fluid condition by the mechanical triturating action of the teeth, jaws, and stomach, as well as by the chemical solvent action of alkaline and acid juices. An observation of the peculiar dyspepsia which so frequently accompanies tubercular disease will satisfy the observer that it depends upon excess of acidity in the alimentary canal, which favours the solution of the albuminous and mineral matters, but is opposed to the emulsionizing of fat. It has consequently been attributed by Dr. Dobell to diminished secretion from the pancreas. In youth the indisposition to eat fatty substances is well marked, and among the ill-fed poor it is fat which is the most costly ingredient of food.[1] In either case it is the non-assimilation of the fatty elements of food, and their diminution in the blood, while the albuminous elements are comparatively in excess, that gradually interferes with nutrition ; the molecular basis of the chyle is impoverished, the elementary molecules so necessary for the formation of healthy blood corpuscles are diminished, the liquor sanguinis consequently is poor in fat and rich in albumen, the entire growth of the constitution, as a result, is affected, and its powers rendered weak ; lastly, when exudations do occur, more especially in the lung, they are of an albuminous character, exhibit slight power of transformation into cells, and only produce that slow abortive nuclear material which is called tubercle. Such is the theory of Phthisis we consider most consistent with all the recognised facts connected with the origin and progress of the disease, the correctness of which is still further supported by what is now known, 1st, of the chemical constitution of the food, and the transformations it undergoes in the body ; 2dly, of the relations which exist between digestion and the working powers of the individual ; and 3dly, as we shall subsequently see, by what experience has taught us of its successful treatment.

II. Symptoms of Phthisis.

From what has been previously said under the head of Morbid Anatomy of Phthisis it must be apparent that the symptoms which it presents will not only have reference to alterations in the functions of the lungs, but to those which may arise from disease in other organs. We must further consider that its onset may be insidious and scarcely perceptible, or on the other hand startling from its violence or acute character ; that its progress may be rapid, slow, or irregular, and its termination ushered in by various phenomena not

[1] See Report by Dr. Edward Smith.

unfrequently of a very complex character. Notwithstanding, to the pathologist who has carefully studied the morbid anatomy, natural progress, and theory of the disease, the symptoms and physical signs of Phthisis will enable him to determine the morbid condition present in the great majority of cases with an exactitude and certainty of which the modern cultivators of medicine may well be proud.

Premonitory Symptoms.—Before any one can positively state that tubercle exists in the lung, there generally occur symptoms indicative of diminished general health, and of deteriorated constitutional vigour. In many cases it is observable in young persons that they are not good eaters, dislike fatty substances, are capricious with regard to food, become thin, pale, weak, and liable to dyspepsia, complain of indigestion and irregularity of the alvine discharges, and to the observant eye are at once recognised as individuals ill nourished and liable to tubercular disease. This condition, however, is often not noticed by the parents or friends, who regard it as only natural to youth, or to the circumstance that they eat so little. On other occasions it creates apprehension and alarm, the physician is consulted, who, however, can detect no pulmonary disease or pulmonary symptom of any kind. If, in addition to the above phenomena, the individual complains of chills, cold feet, occasional perspirations, quick pulse, rendered more frequent at night, the general condition is one highly favourable to the occurrence of Phthisis.

In adult persons the premonitory symptoms are most commonly lassitude, incapacity for following the usual employment, diminution of appetite, with or without indigestion, and a sensible falling off in flesh. Various diseases may manifest themselves, such as gouty or rheumatic attacks, influenza, bronchitis, fever, dysentery, and others, which leave the individual in a debilitated state. There may now come on considerable hæmoptysis, although an examination of the lungs reveals no sign of tubercle ; or an attack of pneumonia may appear, which if treated by lowering remedies may usher in the disease. Occasionally the skin of the face becomes grey, and a haggard and worn expression is communicated to the countenance. Pregnancy and lactation in weak females frequently introduce Phthisis, as, indeed, may everything that calls too strongly for exertion of the vital powers in weak and predisposed persons, or that causes vitiation of the blood. It is in this respect that the recent experiments of Clark, Fox, and Sanderson, previously referred to, indicate how Phthisis may follow suppuration or irritating diseases of texture, and how if occasioned in one organ it may spread to others.

It is when the constitution is thus enfeebled that Phthisis appears in its acute or chronic forms.

Acute Phthisis.—This form of the disease, commonly called "galloping consumption," is generally distinguished not only by its rapid progress, but by the febrile symptoms which accompany it. There are frequent chills, followed by great heat and sweating, red tongue, nausea, loathing of food, vomiting, and diarrhœa. There is a rapid

pulse, at first of good strength, but soon becoming feeble, dyspnœa on slight exertion, cough, profuse expectoration, sometimes tinged with rusty-coloured blood. Occasionally the expectoration is trifling. There is great exhaustion, rapid emaciation, restlessness, and, before death, wandering of the mind and delirium. On percussion one or both lungs exhibit unusual dulness, which rapidly extends and becomes more intensified. It is sometimes most marked at the base. On auscultation there are at first dry, bronchial sounds, and prolonged expiration, which soon pass into moist rattles, loudest with inspiration. The crepitations are now transformed into mucous râles more or less coarse, frequently accompanied with dry bronchial murmurs and pleuritic frictions. The extent of these signs indicates the area of lung-tissue involved, while the amount of increased vocal resonance points out the density of tubercular and pneumonic exudation infiltrating the lungs, or the anfractuous softening and excavations produced.

These acute symptoms occur occasionally in most cases of Phthisis, and indicate the period when exudation is being rapidly deposited in the lungs, or on the pleuræ. In many cases they constitute attacks supposed to be the result of having "caught cold." Then they decline, and are absent for varying periods. The greater the number of these attacks, the more rapid is the progress of the disease; and when they are continuous, it produces that form of it denominated acute Phthisis. Such cases may prove fatal in a period varying from two or three weeks to a few months.

Chronic Phthisis.—In the vast majority of cases the progress of Phthisis is slow, often coming on imperceptibly, and too frequently exciting little attention until it is far advanced. I have known the only daughter even of a medical man slowly pass through all the stages of the disease, the cough and expectoration failing to attract special notice in the family until three weeks before death, when on examination by a physician large cavities were detected. At other times it is ushered in by well-marked disease, such as pneumonia or bronchitis, and in some instances the first symptom observed is hæmorrhage. These different modes of onset in the disease we regard as sufficiently important to merit a separate description.

Gradually-developed Phthisis.—The first symptom which appears is cough; at first, however, so slight as scarcely to attract attention, and attributed to transient exposure to cold, or tickling in the throat. It may be observed, however, to be persistent, and of a dry, hacking character. Sometimes the cough is accompanied with pains in the shoulders, tightness in the chest, slight dyspnœa on exertion, together with all the other symptoms described as premonitory. On percussing the chest no dulness can be detected at this early period; but on auscultation there may frequently be detected feeble respiration under one clavicle, and, during forced inspiration, harshness of the breath murmur, with prolongation of the expiration. After a variable time expectoration follows the cough; at first consisting of transparent, frothy mucus in small quantity, but soon becoming opaque and puru-

lent, and often streaked with a little blood. The cough and expectoration now become gradually increased, and all the other symptoms which have preceded or accompanied them are intensified; the failing appetite is more marked, the quickened pulse and feverish excitement more evident, and the general weakness, falling off in flesh, pallor, and languor make progress. A period, sooner or later, arrives when on careful percussion a sensible dulness may be detected under one clavicle. On auscultation over this dulness, either there is increased harshness of the breath-sound on taking a deep inspiration with prolonged expiration, or a slight crepitation may be discovered during some parts of the inspiratory act. Increased vocal resonance, also, is present over the dull portion of lung. The various symptoms and signs enumerated characterise what many authors regard as the first stage of the disease.

The physical signs now assume marked importance in the history of the case, indicating, in the majority of instances, with great exactitude, the extent of the tubercular deposit, and the changes which it undergoes. The area over which dulness can be detected by percussion gradually extends from the apex downwards, until it occupies one-third, one-half, or even a greater portion of the lung. Dulness may appear at the summit of the other lung, and all the signs observed on the one side may follow on the opposite one. The crepitation on inspiration also extends, and, at first very fine, gradually becomes larger and coarser, until a loud, mucous rattle is established. The vocal resonance, which at first is only slightly increased, becomes louder and louder, until at length decided bronchophony is produced. During the occurrence of these changes in the physical signs, the cough becomes more frequent and prolonged, especially early in the morning, the expectoration is more abundant, and at length consists of dense, purulent masses, some of which sink in water. These also may, from time to time, be streaked with blood, or even slight hæmorrhage from the lungs may occur. There is now generally visible emaciation of the body, considerable debility, indisposition to take exercise, dyspnœa on exertion, and especially on going up an ascent. The tongue is red, often glazed, and occasionally anæmic. There is anorexia and nausea, or the appetite is much diminished, and very capricious. The night sweats are often distressing; there is thirst, quick pulse, and not unfrequently marked fever at night. Sometimes diarrhœa may supervene, which invariably accelerates the progress of the disease. At others there may be various complications, such as attacks of laryngitis, pharyngitis, bronchitis, pleuritis, pneumonia, all of which produce increased weakness, and aggravate the sufferings of the patient. These occurrences characterise what have been termed the second stage of the disease.

The further progress of Phthisis is now characterised by the formation of excavations in the lungs, which are distinguished by loud, moist rattles, passing into gurgling or splashing sounds, if the cavities be large and contain fluid, or by loud, bronchial blowing, and

rarely amphoric breathing, if they be dry. Percussion with the mouth open sometimes elicits a clear tone over such cavities ; at others a peculiar chinking or cracked-pot sound. On speaking there is a shrill vocal resonance, called imperfect pectoriloquy ; and occasionally the words uttered seem to come out of the chest, and strongly strike the ear through the stethoscope, a sign termed perfect pectoriloquy. Together with the signs of a dried cavity are frequently coarse creaking sounds, indicating the existence of chronic adhesions. At the same time dulness, and the other signs audible in the second stage of the disease, are more or less extended over one or both lungs. The cough is now very harassing and prolonged, and often so violent as to occasion vomiting, and it disturbs sleep at night. There is more or less dyspnœa, and occasionally, if the lung be extensively diseased, orthopnœa. The expectoration is greatly increased, consisting of nummular masses of dense, purulent matter, often containing portions of infiltrated lung, which rapidly sink in water. Sometimes it is greenish, ichorous, and of offensive odour. In very chronic cases, on the other hand, with dry cavities, the expectoration is trifling, and brought up with considerable difficulty. Hæmoptysis is now a more common symptom, and may vary in amount from a few teaspoonfuls to twenty ounces, or even more. Such attacks invariably cause great alarm, and produce exhaustion in proportion to the amount of blood lost. The patient frequently complains of pain in the thorax, which in very chronic cases is often severe, ushering in, more or less, flattening of the chest, that may now occur to a greater or less extent. As the disease extends, and the cavities enlarge, the strength of the patient declines, the appetite is lost, and it becomes difficult to eat anything. Hectic fever appears, there is a pink blush on the cheeks, rapid pulse, occasional rigors, profuse sweating at night, and extreme emaciation. Sometimes the vital powers slowly decline, and at length become extinct ; at others, a colliquative diarrhœa appears, which more rapidly closes the scene. These symptoms constitute the third and last stage of the disease.

In the majority of chronic cases the progress of the disease is not uniform, but subject to numerous interruptions, and even long pauses, in which there is decided amendment, with great amelioration and even absence of symptoms. But the physical signs, though they become modified, still indicate the existence of organic lesion. Not unfrequently, however, such pauses and ameliorations are continued for a long period, and in many cases may usher in a permanent arrestment of the disease. In such cases the expectoration gradually ceases, and the cough becomes dry. This, in its turn, becomes less frequent, and at length disappears. Auscultation indicates that the moist rattles are converted into dry blowing or bronchial murmurs. Coarse friction sounds appear, and indicate adhesions and cicatrizations. Dulness on percussion and increased vocal resonance remain, and, although seldom altogether got rid of, become more and more circumscribed, leaving sometimes only a trace behind to indicate the presence of disease. In

severe cases the sub-clavicular regions of the chest are retracted; dense pleuritic adhesions are formed, which circumscribe the movements of the thoracic walls; but healthy respiration is heard in such portions of the lungs as were unaffected. Under such circumstances, although full vigour of body is not restored, life is continued and enjoyed for an indefinite period, and death ultimately caused by circumstances altogether independent of the pulmonary lesion.

Hæmorrhagic Phthisis.—The peculiarity of this form of Phthisis is that it commences with hæmoptysis more or less violent. I have now seen several cases in which individuals who imagined themselves to be in very good health, and in whom, on the most careful inquiry, nothing but some slight dyspepsia or falling off in appetite could be discovered, were suddenly seized with hæmorrhage from the lungs. From that moment their general health began to give way, and Phthisis was developed, of which they died. I remember the case of an extensive sheep farmer in the south of Scotland, who walking home one afternoon—as he thought in the possession of perfect health—was seized in the road with bleeding from the lungs. I saw him a few days afterwards, and failed to detect either from his external appearance, general symptoms, or physical signs, the slightest evidence of pulmonary disease. Nevertheless in a few weeks he became pale and languid, cough appeared, and on his again visiting me a peculiar roughness, or what some call a dry crackling, was distinctly audible at the apex of one lung. He spent the following winter in the south of France; but, notwithstanding every care that could be exercised, he died of Phthisis at the end of three years.

So many cases of this kind have come under my notice, that I have no hesitation in regarding it as a peculiar form of the disease, in which tubercle is deposited in such a manner as in the first instance to induce degeneration and rupture of a considerable-sized vessel in the lung. The loss of blood so occasioned from one or more attacks assists in developing the disease, which subsequently progresses in the usual way. Occasionally such hæmorrhages may occur several times before tubercle deposit has spread so as to be recognisable by physical signs. Not long ago I saw an Australian who for upwards of two years had several such attacks, and who only on reaching this country, in the month of November, when I examined him, had cough developed, with the incipient harshness of respiration.

This form is most common in adults, and is generally fatal, although I have seen a few instances in which, after a time, it was permanently arrested. It is allied to that class of cases in which at any period of the disease hæmorrhage makes its appearance, and is recurrent.

Bronchitic Phthisis.—This form of Phthisis is more common in the young than in adults, and manifests itself in bronchitis, which attacks the apex of one or both lungs. It is a common sequence of severe attacks of influenza, hooping-cough, measles, or other diseases in which the bronchi are affected, in weak persons. They do not readily throw off the pulmonary affection, are very liable to colds;

dyspnœa is readily excited by unusual exertion; they complain of a sense of tightness or constriction about the chest, which, on being examined physically, is quite resonant on percussion; but there is harshness of the inspiratory murmurs on taking a forced breath, with prolongation of the expiration, without increase of vocal resonance. In short, there is slight bronchitis at the apex, which, however, is permanent, or if it disappear for a time shows a great tendency to return. Occasionally there is wheezing, more or less sibilation, and great dyspnœa on exertion, with cough, expectoration, and slight hæmoptysis. For a long time the general health exhibits no further evidence of disease; but at length frequent cough and expectoration appear, weakness, failing appetite, emaciation, and the usual symptoms of Phthisis. In some cases the ordinary physical signs are also manifested; but in others I have known death occasioned without the production of dulness on percussion, increased vocal resonance, or other distinct signs of tubercular consolidation. In such cases, from first to last, bronchitis appears to be the only lesion while the patient wastes away and dies, although on inspection of the lungs afterwards they will be found to contain more or less tubercle. In 1845 I was consulted in the case of a young lady eleven years of age, who, after a violent and prolonged attack of hoopingcough, complained of dyspnœa on exertion and cough. There was no dulness on percussion, and on auscultation there was harshness of inspiration, and slight prolongation of expiration at the apices of both lungs, especially on the right side. Under this affection she laboured for eight years, in all other respects enjoying tolerable health, when the appetite began to fail, purulent expectoration became continuous, and all the symptoms of Phthisis were manifest. She died early in 1855, never having exhibited any of the physical signs of Phthisis, the disease apparently being structurally one of bronchitis and emphysema. On examination after death, however, I found circular patches of miliary tubercle, about three-quarters of an inch in diameter, irregularly scattered through the pulmonary tissue on both sides, together with emphysema.[1] I have since seen several similar cases, and am satisfied that bronchitis developed in weak young persons, especially when it appears at the apex of the lungs, is a frequent prelude and accompaniment of Phthisis, communicating to it a peculiar character, which has frequently led to much error in determining the nature of the disease. This form of Phthisis is allied to all those cases in which bronchitis, in its various phases, constitutes a leading feature of the disease.

Laryngeal Phthisis.—This distressing form of Phthisis is from an early period accompanied by a tickling in the larynx, which seems to be the origin of the cough. The voice becomes weak and hoarse, and not unfrequently there is more or less pain on deglutition. On inspection of the fauces and throat, follicular disease or great dryness of the mucous membrane is common. Sometimes the laryngeal disease

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[1] See the author on Pulmonary Consumption, 2d edit. Case 16, p. 70.

completely masks the pulmonary lesion, causing a hoarse rough murmur on inspiration, which renders the physical signs at the apex of the lung inaudible, so that unless marked dulness is distinguished by percussion, it may be overlooked. Ultimately the voice is lost from destruction of the vocal cords by tubercular ulceration. Deglutition becomes difficult, and vomiting readily excited by reflex actions through irritation of the laryngeal, pharyngeal, and glosso-pharyngeal branches of the eighth pair of nerves. Under these circumstances emaciation makes rapid progress, all the symptoms of ulcerative laryngitis being added to those of Phthisis. (See Laryngitis.)

Pneumonitic Phthisis.—I have now watched a considerable number of cases in which unquestionable Phthisis has originated in an acute pneumonia at the apex of the lungs, which, instead of disappearing in the usual way, has become chronic. Under such circumstances the dulness on percussion and bronchophony remain, the summit of the lung is consolidated, the general health, instead of rallying, remains weak, cough and expectoration become troublesome, while loud mucous and gurgling rattles are gradually formed in the lung, indicating the existence of cavities. Sometimes the consolidated lung remains latent for a considerable time, the patient in vain endeavouring to restore his original strength. Then an attack of hæmoptysis has occurred, which induces him to visit a physician, and it is discovered that the lung is consolidated, and all the signs of Phthisis are more or less apparent. Discussion has taken place as to whether such cases should be denominated chronic pneumonia or Phthisis. In my opinion there is no difference between them. The exudation of the pneumonia degenerating, and not being absorbed, is transformed into tubercle, causing softening, ulceration, and destruction of the lung, in exactly the same way as if Phthisis had been developed from tubercle at the commencement. I have also seen survivors from this form of the disease with flattening of the chest, as in ordinary chronic Phthisis. It must not be overlooked either that intercurrent attacks of pneumonia are very frequent during the progress of Phthisis, and that at all times the two diseases exhibit a marked tendency to run into one another. This circumstance confirms the truth of the pathology previously given, and unequivocally proves that tubercle is only a low type of exudation from the blood. In healthy persons such exudation is transformed into pus, and rapidly disappears, whereas in individuals who are weak, and whose vital power is low, this process is more or less interfered with, is prolonged, and in extreme cases terminates in Phthisis. This view has recently been adopted by Niemeyer, who is one of those who proposes to call Phthisis Pulmonalis a chronic pneumonia, in the propriety of which, as applied to all its forms, I cannot concur.

Complications.—Tubercular disease of the lungs is necessarily associated with every lesion occasioned by inflammation and tubercular exudation of the textures of the organ. Indeed it may be said to

be made up of exudation disorders, acute or chronic, affecting the air vesicles, bronchi, fibrous tissues, and serous coverings of the lungs. Hence the various symptoms of laryngitis, bronchitis, emphysema, hæmorrhage, pneumonia, and pleurisy are more or less mingled together, may supervene on each other, and occasionally, as we have seen, be so predominant and permanent as to give peculiar characters or forms to the disease. Occasionally pleuritis gives such a character to Phthisis, occasioning local acute or stitching pains. Tubercular cavities in the majority of cases induce thickenings and dense adhesions between the pleuræ, but sometimes they may burst or ulcerate through the pleuræ, where there is no adhesion, causing pneumothorax, associated or not with more or less empyema.

In addition, however, to these lesions of the chest, Phthisis may be associated with tubercular deposits occurring in other organs, in which case a train of symptoms will arise dependent upon the local lesion, wherever that may be. Of these the most common, and the most to be dreaded, is tubercular ulceration of the intestines, inducing colliquative diarrhœa, and perhaps perforation of the gut, with fatal peritonitis. In the young, also, we may find the disease associated with various tubercular or scrofulous diseases of the osseous texture, and sometimes of the brain or its membranes. It would exceed our limits to enter upon the innumerable complications which in this manner may arise ; all that it is necessary to say is, that there is no tubercular disease of any organ or tissue which may not be found sometimes associated with Phthisis, and which, contributing its own special symptoms to the pulmonary ones, increases the general disease and downward progress of the patient.

Besides this class of affections, there are others of importance. It is by no means uncommon during the progress of Phthisis to find persons complaining of puffiness of the feet, or face, and on examination of the urine it will be found to contain albumen. In short, one of the forms of Bright's disease may develop itself, and usually that now recognised as the waxy form. The liver also may enlarge, and add to the distress of the patient by its pressure and bulk. Such increased growth of the hepatic organ will also generally be found to be dependent on a waxy transformation of its cells and vessels. The spleen may undergo a like alteration, although its enlargement is more rare. Pericarditis and other inflammatory diseases may occur—occasionally gout or rheumatism. Cancerous disease, it is now known, may be associated with Phthisis, but it is an occurrence of extreme rarity. In chronic cases the practitioner must be prepared to meet with a variety of other complications, which, though they may bear no essential or constant relation to Phthisis, render the disease more distressing and fatal should they occur.

III. DIAGNOSIS OF PHTHISIS PULMONALIS.

It has been previously pointed out that Phthisis is preceded by pre-monitory symptoms, which indicate diminished health, weakness, or imperfect nutrition of the individual. This condition has been spoken of by some writers as constituting a pretubercular stage of disease. All that can be said, in a diagnostic point of view, of this state of health, is that in young and delicate persons it should occasion much anxiety, as it may, or may not, terminate in Phthisis, and that it should demand great watchfulness and frequent careful examination, in order that the first positive signs of the disease may be detected.

ACUTE PHTHISIS.—The diagnosis of this form of the disease is ex-ceedingly difficult, as all the symptoms and signs are identical with those of an acute inflammation of the lungs. It is only by careful observation of the premonitory symptoms, the existence of a marked hereditary taint, the amount of emaciation as compared with the extent of local disease, the continuity of the fever, and the rapid formation of cavities, that we are at length able to pronounce with confidence as to the presence of acute Phthisis. In all its essential features the attack is similar to acute pneumonia of the apex, from which in its earliest stages it cannot be separated. As the disease pro-gresses, however, the excessive exhaustion and breaking down of the lungs establish the nature of the affection, while its rapid progress and the continued fever too certainly indicate its acute nature. In the present day the extreme difficulty of diagnosis is fortunately not of so much importance as it used to be, when such symptoms led to bleeding, and an antiphlogistic treatment. In the course of chronic Phthisis similar symptoms may arise, either from fresh exudation of tubercular matter, or from intercurrent attacks of pneumonia or pleurisy, communicating to the disease for a time an acute character.

CHRONIC PHTHISIS.—In this, by far the most common form of the disease, it is of the greatest consequence to determine its commence-ment by the conjoined methods now in vogue. Its progress is capable of being recognised with considerable certainty, and the means at our disposal for doing this may be considered under the heads of Pulmonary Symptoms, Pulmonary Percussion, Pulmonary Ausculta-tion, Microscopical Examination of the Sputum, and Altered Changes in the Form and Movements of the Chest.

Pulmonary Symptoms.—The earliest symptom is cough, which, at first short and dry, resembles the ordinary effort at clearing the throat. Sometimes it is attributed to the chest, but more commonly is thought to arise from dryness or tickling in the throat. Such a cough too frequently excites little attention, although its persistency and defiance of ordinary remedies communicate to it a grave charac-ter. After a time the cough is followed by expectoration, at first of

a thin mucous fluid, which, however, soon becomes thick and opaque, or is slightly streaked with blood. There is now occasionally felt a tightness or constraint, on taking a deep breath, under one clavicle, which, as the disease progresses, becomes painful, especially on coughing. This cough and expectoration, more particularly when they follow the premonitory symptoms, and are developed in the manner described, are highly characteristic of Phthisis. In the subsequent stage of the disease, the cough becomes more frequent, harassing, and long-continued. The tickling in the throat may excite vomiting. The expectoration is more abundant and prevalent, frequently tinged with blood, and forms distinct masses (nummular sputa) generally indicative of excavations, and may be so heavy that, instead of floating, it sinks in water. Lastly, it may contain masses of indurated matter, composed of portions of tubercular lung, or, in very chronic cases, fragments of cretaceous or calcareous matter. Early hæmoptysis, as we have seen, is highly diagnostic of Phthisis, and should always excite grave attention. Should it be soon followed by or mixed up with the other symptoms, the diagnosis is considered more certain.

Pulmonary Percussion. — When miliary or infiltrated tubercle occupies a certain number of the air vesicles, careful percussion above or under one clavicle elicits slight dulness of the pulmonary note, especially well marked when compared with the clear note on the opposite side. As it is seldom that the disease commences at the apices of both lungs at once, this sign is one of great value, and indicates very positively, not only the existence, but very frequently the extent of the disease. The greater the dulness or flatness of tone, the more solid is the portion of lung struck ; and the further over the chest, anteriorly and posteriorly, the dulness can be produced, the greater is the amount of pulmonary tissue involved. It should not be overlooked, however, that occasionally the disease exists equally on both sides, when diagnosis by means of percussion is always difficult. In the earlier stages, indeed, it is then impossible, and in the later stages, even with large cavities on both sides, I have known the percussion note so equal and clear as to mislead the careless observer. Sometimes also, though the lung be greatly condensed, an amount of emphysema anteriorly communicates clearness on percussion: hence the lung should always be examined posteriorly as well as anteriorly, in order to avoid error.

On percussing the chest in cases of Phthisis with the mouth open, there is sometimes elicited a peculiar noise, called by Laennec the *bruit de pot fêlé*, or cracked-pot sound, which he thought was diagnostic of a cavity. But I have found this noise could also be produced in cases of pneumonia, in pleurisy with effusion, and even in several healthy chests. Moreover it is often absent when pulmonary cavities are unquestionably present, and cannot therefore be considered as diagnostic of their presence, unless it be co-existent with other symptoms and signs of Phthisis. When present, it seems to indicate

either healthy lungs, with very elastic thoracic walls, or else increased density mingled with confined or compressed air in the thorax. In either case, on striking the chest smartly, the air beneath is forcibly ejected through the bronchi and trachea, producing vibrations which occasion the peculiar sound.[1]

Pulmonary Auscultation.—The first sounds audible with the stethoscope are prolongation of the expiratory murmur and slight harshness, or a wavy interrupted character communicated to the inspiratory murmur. These signs, if clearly marked under one clavicle, following the premonitory symptoms, and accompanying persistent hacking cough, can leave no doubt that tubercle is actually present, and the disease pronounced. It frequently happens, however, that these signs are so indefinite that, although we may suspect, we hesitate to speak confidently. In all chronic organic diseases there must be a period so nicely balanced between health and disease—in which the altered texture is so slightly altered—that our senses are incapable of appreciating any alteration that may be produced. It is in such cases that everything which enables us to determine such delicate signs with greater exactitude becomes valuable, and I have no hesitation in stating that the differential stethoscope of Dr. Scott Alison has here afforded me the greatest assistance. In several delicate young persons, in whom when every precaution and care has been employed we fail to discover any alteration in the pulmonary sounds, an increased intensity in the sound of the carotid artery below the clavicle has afforded valuable indications. It is at this early and uncertain period of the disease that the greatest skill in auscultation and diagnostic powers are required in the physician.

As the disease advances, the prolonged expirations and harsh inspirations become more marked, and at length a decided increase in the vocal resonance of the affected side is audible. This indicates considerable condensation of the apex of the lung. If the disease progresses, slight crepitation is audible, at first at the termination of a forced inspiration, and gradually it occupies the whole of that act. This is diagnostic of tubercular softening. The fine moist rattle now becomes evident, and the increased vocal resonance louder, until it amounts to bronchophony. The auscultatory signs also extend in area over the chest, preceding the dulness on percussion, and generally appearing in the order in which they were noticed over the apex. At length the crepitation passes into mucous râle. This in its turn becomes coarser and coarser, indicating the existence of greater softening, and even of cavities. As these enlarge, gurgling and splashing sounds are heard, especially on coughing, and the increased vocal resonance becomes pealing, and imperfect or perfect pectoriloquy is present. These latter sounds are diagnostic of a cavity or cavities. The sounds heard over these vary according to their size, contents, and the condition of the walls. If large, with rigid walls, and partly filled with

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[1] See the author on Pulmonary Consumption, &c., Diagnostic Value of the Cracked-pot Sound, p. 108.

fluid, and partly with air, tinkling or metallic sounds may be heard on coughing or speaking. If altogether dry, amphoric or blowing noises may be distinguished. These last, if persistent, indicate that the secretion of pus is arrested, the softened tubercle got rid of, and contraction and cicatrization possible.

When in chronic cases of Phthisis dry blowing, combined with friction sounds, can be determined at the apex, it points out that adhesion and contractions of the tuberculated pulmonary tissues are taking place. If absence of respiratory murmur exist, it may depend on pleuritic effusion, when dulness on percussion and increased vocal resonance, or ægophony, will determine the nature of the lesion. But it may be accompanied by resonance on percussion, with a brazen, hollow, or metallic sound on coughing or a forced inspiration; in which case there is pneumo-thorax, and the tubercular cavity has formed a communication with the pleura.

In retrograde Phthisis, the auscultatory signs disappear in the inverse order to that in which they appear. The moist sounds become dry, and these last diminish in intensity and extent. Friction noises and dry bronchial murmurs are heard, with prolonged expiration, wheezing, and sonorous rhonchi indicative of rigid bronchial tubes, conjoined with more or less emphysema. The area of dulness gradually diminishes, but a condensed mass in the lung generally remains for years at one or both apices, giving rise to harsh respiratory murmurs and increased vocal resonance, constituting strong evidence to the judicious observer of the diseased changes through which the lung has passed.

Microscopical Examination of the Sputum.—The sputum of phthisical patients, in the great majority of cases, may be found to contain, under the microscope, fragments of the areolar and elastic tissues, derived from disintegration of the lungs. They not unfrequently present circles and half-circles, indicative of the form of the air vesicles, and, when present, offer the most positive proof of pulmonary ulceration. Van der Kolk, of Utrecht, was the first to point out that such fragments might be seen with the microscope, at the commencement of the disease, long before percussion or auscultation gave any positive signs of its existence. Although such examples are rare, I am satisfied that they do occur, and that the microscopical examination of the sputa under such circumstances enables us to arrive at a clear diagnosis when otherwise there would be great doubt. Drs. Andrew Clark and Fenwick have confirmed this fact by their researches into the structure of phthisical sputum. The latter physician has pointed out that the examination is much facilitated by first liquefying the sputa with a solution of caustic soda, when the fragments of lung tissue are precipitated, and their amounts as well as character readily estimated.

Altered Changes in the Form and Movements of the Chest.—As Phthisis advances, a distinct flattening and sinking in of the thoracic walls below the clavicle may be observed, generally coincident with the

formation of cavities and loss of lung substance, of which it is diagnostic. An alteration in the movements of the affected side may be seen even earlier, and may be roughly ascertained by spreading the fingers of both hands like a fan over the two sides of the chest, and bringing the thumbs together at the middle of the sternum. On a forced inspiration, it may thus easily be seen that the thumb corresponding with the affected side moves less. The amount of this movement can be ascertained with great exactitude by means of the stethometer, and compared with that on the opposite side.

In addition to the symptoms and signs referable to the chest, there must not be overlooked a variety of circumstances which in conjunction with these will materially assist the diagnosis. Among these are the preceding premonitory symptoms; the continued impaired appetite and disordered digestion; the augmenting languor and debility; the hectic night-sweats; lustrous eyes; the hopefulness and imaginative intellect; and even the alternations of the disease from better to worse, all of which are more or less characteristic.

Much has been written concerning what is called the differential diagnosis of Phthisis, and the means of distinguishing it from other diseases of the chest. But the truth is that a Phthisis necessarily implies the existence of almost every lesion of the lung, the tubercular exudation giving rise to or being accompanied by congestions and inflammations of the pleuræ, bronchi, and pulmonary parenchyma, with all their local signs and general symptoms. Pulmonary hæmorrhage and abscess are common. Emphysema, though seldom present in its advanced stage, so as to alter the form of the chest, is common in limited portions of the lung near chronic and retrograde tubercular deposits. Any lesion whatever, occurring at the apex of the lung in a young person labouring under the premonitory symptoms we have described, must be regarded with suspicion. In adults, an acute pneumonia at the apex may go through its natural progress, and leave no trace behind. But if it becomes chronic, a Phthisis may be the result. Indeed there are many cases in which a chronic pneumonia of the apex and Phthisis Pulmonalis may be said to constitute the same disease. Cancer of the lung is a disease of advanced age; the dulness on percussion is more marked, the tubular respiration and bronchophony are much greater, and moist rattles are scarce or absent. Expectoration is trifling, and, when present, unlike that of Phthisis; sometimes it resembles currant jelly. The emaciation, night-sweats, and general aspects afford little assistance. A dilated bronchus, independent of Phthisis, is rare, but when present is often associated with bronchitis and asthmatic symptoms, while the physical signs of the cavity are generally best marked at the posterior and middle regions of the chest, rather than at the apex. In advanced cases a pleurisy with effusion or a pneumo-thorax may occur, when the physical signs distinctive of each will readily establish the diagnosis.

The great difficulty is to detect Phthisis at its first appearance, and hence every circumstance that can throw light on its history at

this period is important. According to Dr. Ringer, the heightened temperature of the body, as determined by the thermometer, indicates the deposition of tubercle for several weeks before physical signs are developed. It is true that a similar increase of temperature occurs in a few other diseases, such as typhoid fever or rheumatism, but their symptoms are readily separable from those of Phthisis. This new method of recognising the disease at an early stage requires more extended observation before it can be generally adopted. The subsequent progress of Phthisis admits of being followed by the physician cognisant of its morbid anatomy, and well skilled in auscultation, not only with certainty, but in the majority of cases with a degree of exactitude that must be regarded as highly honourable to the progress of medicine in modern times.

IV. Prognosis of Phthisis Pulmonalis.

Phthisis Pulmonalis, up to a comparatively recent date, was not only regarded as a very dangerous disease, but as one which was uniformly fatal. This idea was supported by the circumstance that before the general introduction of physical diagnosis it was not clearly detectible until it was far advanced, while the merely palliative treatment then in vogue was anything but favourable to recovery. If, notwithstanding, a case here and there did ultimately get well, medical men were more disposed to accuse themselves of an error in diagnosis than doubt the correctness of so general a dogma as the incurability of consumption. Even when at length morbid anatomy unequivocally demonstrated the possibility of tubercular cavities cicatrizing, and of individuals afterwards attaining an advanced age, such an event was regarded as one of extreme rarity, and as occurring altogether independently of treatment. "No fact," says Andral, "demonstrates that Phthisis has ever been cured, for it is not art which operates in the cicatrization of caverns; it can only favour this at most, by not opposing the operations of nature. For ages remedies have been sought to combat the disposition to tubercles, or to destroy them when formed ; and thus innumerable specifics have been employed and abandoned in turn, and chosen from every class of medicaments." Even Louis, in his admirable work, while admitting that a cure might rarely take place, points out that in such cases the disease must be limited and the result fortuitous. Hence the admitted occasional recoveries in no way interfered with the general view entertained of the unfavourable prognosis of this malady, or stimulated medical men to replace a palliative by a curative treatment.

At present, so far from Phthisis being considered to be uniformly or even generally fatal, it is admitted that treatment can in a great majority of cases prolong life, whilst in many, the number of which is annually increasing, a complete and permanent cure may be effected. This revolution in our prognosis of the disease is owing—1st,

to the facts arrived at by morbid anatomy; 2d, to a more perfect theory or pathology of the disease; and 3d, to the discovery of cod-liver oil as a remedy.

1. The careful post-mortem examinations now made with such regularity in our large hospitals have demonstrated the frequent occurrence of old condensations, cicatrices, and calcareous concretions at the apices of the lungs in persons of advanced age who have died of other diseases. In 1845, I pointed out that in the Royal Infirmary of Edinburgh they occurred in the proportion of from one-fourth to one-third of all the individuals who died after the age of forty. Roger and Boudet had previously shown that at the Salpêtrière and Bicêtre hospitals in Paris, amongst individuals above the age of seventy, they occurred in one-half and in four-fifths of the cases respectively. There can be no doubt that these cicatrices and concretions indicate the healing and drying up of cavities and softened tubercular matter at some previous period in the life of the individual, and the consequent spontaneous cure of the disease in a considerable number of persons.

2. The careful examination of tubercle by means of the microscope demonstrates that it neither originates in nor gives rise to cell formations, but that it consists of an exudation of the blood rendered feeble in vital power by impaired nutrition, and especially by deficiency of primary molecules of fat in the blood.[1] Hence the encouragement given to our efforts in stimulating the nutritive functions, and especially assisting in the increased assimilation of an easily digestible oil, whereby, while the tissues generally are supplied with formative material, the tubercular matter has time to degenerate and be absorbed; so that any cavities which have been produced may cicatrize. Attempts at cure in this direction have been so eminently successful as to influence our prognosis in a marked manner.

3. It is very much to be doubted, however, whether this pathology would ever have been arrived at, or if it had, whether a successful treatment could ever have been established, unless the therapeutical properties of cod-liver oil had been recognised. This animal substance is easily assimilated, is not purgative, and meets all the indications required, while experience has demonstrated that it restores to the emaciated body the nutritive elements it so much requires, and enables it to triumph over the disease. It can no longer, therefore, with truth be considered that Phthisis Pulmonalis is that *opprobrium medicinæ* it was formerly considered. Nor should certain charitable institutions any longer refuse to admit such cases on the ground of their incurability.

In my work on Pulmonary Consumption[2] will be found full details of the arrest of the disease in its most advanced stage, the individuals not only being still alive, but having enjoyed excellent health since

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[1] In making this statement I am fully aware of the observations and arguments of Virchow and his followers, but which, for the reasons previously given, I regard as not only inconsistent with histological and pathological research, but as especially opposed to all we know of clinical facts in modern times.

[2] Pp. 152 et seq.

their recovery, for periods varying from ten to twenty-five years. To the list of cases therein given I could now add many more. Twelve similar cases were recorded by Dr. Quain in 1852,[1] and many others may be found scattered in the works of different authors, and in the practice of individual medical men. There can be little doubt that could they be collected, it were easy to prove that such examples, instead of being few and far between, are much more numerous than is generally supposed. It is very difficult, however, to watch for many years in succession the progress and termination of chronic Phthisis; and in hospitals this difficulty is increased, as the patients on getting better go out long before the disease is even permanently arrested. All attempts to induce medical men to unite and record their experience on this or any other great question involving the prognosis or treatment of disease have hitherto failed. We are, therefore, limited to the conscientious efforts of individuals in our attempt to elucidate this question, which cannot be expected in a matter of such magnitude and importance to be, at present, of any great avail. Among these, however, I have great pleasure in referring to the accurate method in which Dr. Pollock has recorded his ten years' experience at the Brompton Consumption Hospital.[2] Were such method and care more uniformly practised by hospital physicians, and extended over more lengthened periods, many of the unsolved problems connected with this subject might be elucidated. I confidently look to the future, as affording means for demonstrating the ratio and conditions under which the prognosis of Phthisis may be determined. In the meantime, I can only express my conviction that its permanent arrestment and cure are, by judicious treatment and hygienic management, becoming every day more frequent and more widely extended.

In reference to the prognosis of individual forms or cases of Phthisis, we must regard acute Phthisis as generally fatal. The difficulty here lies in the diagnosis. Once recognised, however, the persistency of intense fever, with rapid emaciation and formation of cavities, give us little hope of a favourable termination.

In the earliest periods of Phthisis, the prognosis should be very guarded, but on the whole encouraging. As a general rule, the more slowly it advances, the less fever and emaciation, and the better the appetite, the more probability exists of an arrestment.

In the second stage, the favourable symptoms are limitation of the disease to one lung, dulness not extensive, and not increasing rapidly; no persistency of moist rattle; expectoration moderate; fever trifling; emaciation not great; capability of taking nourishment and a certain amount of exercise. The unfavourable symptoms are continuous fever, quick pulse, hæmoptysis repeated, profuse expectoration, rapid softening of the tubercle, and its deposition in both lungs; bad appetite and impaired digestion; increasing emaciation; profuse diaphoresis, and the existence of unfavourable complications.

[1] Lancet, pp. 487 et seq.
[2] The Elements of Prognosis in Consumption. London, 1865.

In the third stage, the favourable signs are the existence of a cavity in one lung; gurgling or other moist rattles occasionally disappearing, and the excavation becoming dry, with blowing sounds; gradual flattening of the sub-clavicular space, while the other parts of the chest move freely. Further, the disease in the opposite lung absent, or if present slight, without a tendency to extend; coarse friction sounds over the cavity; and a general tendency to concentration, density, and fixity of the lesion. The favourable symptoms accompanying these local changes are, a tranquil pulse, no fever or sweating, emaciation checked, tolerable appetite, and capability of digesting nutriment, diminished cough and expectoration, power of taking more exercise and gaining flesh, and absence of complications.

On the other hand the unfavourable symptoms are the converse of these, especially cavities on both sides, loud moist and gurgling rattles, increasing dyspnœa, profuse expectoration, especially of greenish or ichorous matter, extreme emaciation, anorexia, nausea, vomiting and incapability of retaining or digesting nutriment, profuse diaphoresis and quick pulse, fever, and restlessness at night. If now any serious complication arises, more particularly continued diarrhœa, albuminous urine with œdema of the feet or ankles, laryngitis, or pneumo-thorax, &c. &c., death is not very distant. It is very rarely that hæmoptysis proves fatal, but should it occur profusely when weakness is extreme, death may be immediate.

V. Treatment of Phthisis Pulmonalis.

The treatment of Phthisis Pulmonalis, up to a recent period, has been too much governed by a desire to relieve symptoms—in other words, has been more palliative than curative. Unfortunately the remedies useful for the former purpose are altogether incompatible for the latter, and ultimately even fail to relieve the functional derangements to which they are directed. A study of the pathology of the disease has led us to the conclusion that Phthisis is dependent,— firstly, on impoverishment of the blood; secondly, on exudations into the lung, which assume a tubercular character; and thirdly, on destruction of the lung, owing to the successive deposition and softening of these. It follows that, instead of endeavouring to relieve cough or favour expectoration, our chief attention should be directed to improve the faulty nutrition, to cause absorption of the tubercular exudation, to arrest the ulcerative process; and, lastly, prevent a recurrence of the disease. The special treatment required in individual cases should be made subordinate to these great ends—at all events should not be opposed to them. We shall therefore consider the treatment as general and special: the first directed to favour the removal of the pulmonary lesion; the second to check occasional symptoms and complications.

General Treatment of Phthisis Pulmonalis.—The great indication in the treatment of Phthisis Pulmonalis should be to

improve the nutrition of the economy. This does not merely consist in increasing the quantity and improving the quality of the food, but in employing all those means which shall secure—1st, an appropriate diet; 2d, causing its assimilation, and the formation of good blood; 3d, securing the proper purification of this by the atmosphere; 4th, seeing that a proper demand for the addition of new matters to the tissues is created by sufficient exercise; and 5th, that the effete matters be properly excreted from the economy by the emunctories. All these processes are comprehended in the function of nutrition. We shall most concisely convey our ideas as to the best means of increasing nutrition in phthisical cases, under the distinct heads of Diet, Cod-liver Oil, Pure Atmosphere, Climate, Exercise, and Bathing.

Diet.—One of the leading symptoms in cases of Phthisis is the diminished, capricious, or disordered appetite, and power of taking food. It is true that many cases persistently assure you that they eat heartily, but on careful inquiry they will admit their appetite is easily satisfied, or that they are small eaters. Even the friends sometimes assert that the patients eat as usual, that they have observed no change and so on, the fact being that the nutritive matter actually taken into the economy is far less than it ought to be. So little observation and attention do those affected exhibit concerning their own cases, and so anxious do they appear to represent every circumstance in the most flattering point of view, that it is far from uncommon for them to declare themselves as constantly getting better, up to the moment of their death. I have frequently pointed out to my clinical pupils that, in the reports of these cases taken down by the clerk at the bed-side, in answer to questions, it has been recorded day after day that the appetite is better and better, while the patients are visibly getting more emaciated, more weak, and at length die. Among the poor and half-starving population, it frequently happens that it is not the appetite or desire for food that fails, so much as the food itself. The result here, however, is the same, viz. that the body is not sufficiently nourished, that the tissues disintegrate more rapidly than they can be supplied with new substance, and that the blood is deficient in what is so necessary for supporting health.

In all cases of Phthisis Pulmonalis the diet should be generous, consisting of boiled milk, cream, eggs, butter, toasted bread, and all kinds of animal food, and farinaceous puddings. Acid substances and drinks should be as a rule avoided, the tendency to dyspepsia from too much acidity being generally present. After dinner a glass or two of generous wine (sherry) or two or three glasses of sound claret (not acid) may be indulged in. As much variety as possible should be secured, and every pains taken by good cooking and superior quality of the viands to tempt the weak and capricious appetite. As to quantity, I have never seen any necessity for limiting it. The only difficulty is to take enough; the which once accomplished, amelioration in all the symptoms may be confidently predicted. It should be remembered, however, that mere eating and loading the stomach, without a proper digestion

and assimilation, can be of little benefit. In this respect individuals differ, some doing best with two or three meals a day, whilst others find that eating more frequently, but less at a time, answers better. In nothing is the constant attendance of a judicious medical man more serviceable than in watching the effects of diet, and observing from its influence on each individual case how it should be regulated.

When fever runs high, the pulse is quick and the tongue furred, there will naturally be no disposition to take solid food. Under these circumstances we should take care that nutritious drinks are regularly administered, especially beef-tea and milk, and seize the earliest opportunity of returning to a more substantial diet. Many may think that in most acute cases, or during an intercurrent attack of pneumonia, these rules should be departed from. Formerly, indeed, antiphlogistics and the local application of leeches were employed; but it has now been satisfactorily demonstrated that even in acute cases of pneumonia itself, in vigorous constitutions, such practice is injurious:[1] how much more, then, would it be so in cases of Phthisis? Inflammations are now recognised to be diseases of weakness, and we feed them as we do fevers, with the most marked success. When therefore attacks of either supervene during the progress of Phthisis, so far from doing anything to diminish the strength of the economy, the most anxious care will be required on the part of the practitioner to counteract, by all the support he can administer, the future exhaustion of his patient.

An increase in the quantity and improvement in the quality of the food may frequently be observed to benefit cases of Phthisis, especially among the half-starved poor; the more so if associated with change of scene, active exercise, or varied employment. The treatment practised at the commencement of this century by Dr. Stewart, of Erskine, near Glasgow, which consisted in freely administering beefsteaks and porter, and causing exercise to be taken in the open air, excited considerable attention in its day by the success it occasioned. I have been informed that in America the consumptive patient, by eating the bone marrow of the buffalo on the prairies, is at length enabled to hunt down the animal. I have known several young men on large sheep farms in Australia cure their tubercular lungs by eating fat mutton and galloping about on horseback. Whenever food rich in fat can be tolerated by the stomach, it will produce like effects, and hence the occasional value of bacon, pork chops, caviare, suet, yolks of eggs, and the produce of the dairy, such as milk, cream, and butter.

Cod-liver Oil.—All good food must consist of a proper mixture of albuminous, fatty, and mineral principles. The two former, holding the third in solution, after being prepared by the digestive fluids form a molecular fluid—the chyle—out of which the blood is formed. In Phthisis, however, the process of chylification is impaired; the fatty constituents of the food are not separated from it and assimilated, or they are deficient, as very commonly results from a dislike to fatty substances. In either case, the blood abounds in the albuminous

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[1] See the author's Treatise on the Restorative Treatment of Pneumonia, 3d edit. 1866.

elements, and when exuded into the lungs, as we have seen, forms tubercle. To induce health, it is necessary to restore the nutritive elements which are diminished, and this is done directly by adding a pure animal oil to the food. By so doing, we form richer chyle and better blood; we restore the balance of nutrition, which has been disturbed; respiration is again active in excreting more carbonic acid gas; the tissues once more attract from the blood the elementary molecules so necessary for their maintenance; the entire economy is renovated, so that while the histogenetic processes are revived, the histolytic changes in the tubercle itself also are stimulated, and the whole disappears. We have previously seen that food rich in fat will occasionally produce these effects, but then the powers of the stomach and alimentary canal must not have undergone any great diminution. In most cases, however, the patient is unable to tolerate such kind of food, which is not digested. Under these circumstances, cod-liver oil is directly indicated, by giving which we save the digestive apparatus, as it were, the trouble of separating fluid fats from the food. By giving the oil directly in quantity, a large proportion of it enters the system, unites with the albumen, and thereby forms the molecular basis so essential for the chyle. Since the days of Liebig, chemists have generally supposed that albumen forms the basis of the tissues, and is a flesh-former, while fat is necessary for respiration, and by its decomposition furnishes heat. An unacquaintance with histology is the cause of this error, fat being demonstrably necessary for the development and support of muscle and of every tissue. This has recently been further shown by the investigations into the diet of labourers by E. Smith, the feeding of animals by Lawers and Gilbert, and the experiments of Haughton, Frankland, Fick, and Wislicenus. Hence the universal craving and necessity for fat by the vigorous and working man, whilst a dislike to it is a strong symptom of inherent weakness, and an incapability of assimilating it the chief cause of tubercular disease.

It was in the years 1840 and '41 that I found cod-liver oil used very generally in the German hospitals in all scrofulous and phthisical cases. In England it at one period had been employed in Manchester, at the beginning of the century, by Drs. Kay and Bardsley, in rheumatism, but had fallen into neglect. In the hospitals of Heidelberg and Berlin, I watched with great care the effects of the oil in several cases of Consumption, and satisfied myself of its remarkable powers as a nutrient, under circumstances which in British hospitals would have been attended with little hope. In the autumn of 1841, therefore, I published a monograph containing an account of what was then known of this substance, and recommended it especially to my countrymen, both from theoretical and practical grounds, as a valuable remedy in Phthisis.[1] The first physician who tried it in the Royal Infirmary of Edinburgh was Dr. Spittal; but so little were druggists

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[1] See the author's treatise on the Oleum Jecoris Aselli, &c. 1841; also with Appendix, 1847.

acquainted with the oil, that I found on visiting his wards that all
the patients were taking linseed oil. The same mistake had pre-
viously occurred to Rush, in Berlin. I was therefore obliged to
get it made expressly, which, after a time, was done by the Messrs.
Parker and Co., oil merchants, Leith-walk, who for many years
made the purest cod-liver oil in Great Britain, which they sent
over the country at the moderate rate of 16s. a gallon. When in
the course of time it was asked for in London, Mr. Jacob Bell, the
eminent druggist in Oxford-street, caused a very pure oil to be
made from the livers of the cod, which, however, was so expensive,
that he dispensed it at the rate of half-a-crown an ounce. I
was consequently written to by numerous persons in London and
elsewhere, and was thus the means of causing hundreds of gallons
to be distributed by the Messrs. Parker to all parts of the country.
Gradually, its value was generally appreciated throughout Scotland,
and was extending in England, when it was tried in the Brompton
Consumption Hospital of London. In 1849, Dr. Williams pub-
lished a paper, in which, from extensive trial of the remedy, its
value and mode of action were confirmed, a result still further sup-
ported by the Report of the Brompton Consumption Hospital, pub-
lished in 1851. Since then the employment of cod-liver oil in
Phthisis has been almost universal, and has contributed in no small
degree to remove that hopelessness and despair with which the treat-
ment of the disease had been previously accompanied. In 1841, it
was unknown in our druggists' shops, except here and there, where it
was kept in small quantities for the use of tanners, who, curiously
enough, had discovered that it possessed far superior power to all other
fatty substances in penetrating and softening leather. At that time
the eminent Edinburgh druggists, Duncan and Flockhart, did not
dispense one gallon in the year, whereas at present they dispense
between six and seven hundred gallons annually.

A most extensive experience has now amply confirmed the opinion
I published regarding it thirty years ago—viz. "That no remedy has
so rapidly restored the exhausted powers of the patient, improved the
nutritive functions generally, stopped or diminished the emaciation,
checked the perspiration, quieted the cough and expectoration, and
produced a most favourable influence on the local disease. Many
individuals presenting the emaciation, profuse sweats, constant cough
and expectoration, as most prominent symptoms, with a degree of
weakness that prevented their standing alone, after a few weeks' use
of it, are enabled to get up with ease and walk about, with a visible
improvement in their general health, and an increased amount of
flesh." Thus it must be regarded as an analeptic ($\dot{\alpha}\nu\alpha\lambda\alpha\mu\beta\dot{\alpha}\nu\omega$, to
repair) or general restorative, being digestible where no other kind of
animal food can be taken in sufficient quantity to furnish the tissues
with a proper amount of fatty material. It is not by a chemical so
much as by a histological process that the result is produced.

By some, however, it is supposed that the superiority of cod-liver

oil over other fatty substances is owing to the iodine, bromine, resin, and other medicaments it contains. But the quantity of these drugs in cod-liver oil is very minute, and it has been abundantly proved that no combination of them given internally has any effect on the progress of Phthisis. Hence the idea of giving a watery extract of cod-liver oil, when the oil cannot be taken, appears to us to be erroneous in theory, and unlikely to succeed in practice. On the other hand, there are so few persons who cannot take the oil when it is absolutely necessary, that such preparations need be very seldom employed. I have known many individuals who prefer the brown and apparently nauseous to the light and comparatively pure oil. In all cases, that kind of oil is best that is most readily tolerated by the stomach. Those who at first express dislike to the remedy, by a little perseverance may be made to take it readily; if not, they should try whether it be retained best immediately before, immediately after, or in the intervals of meals. The crunching a biscuit, or a lump of sugar on which there has been placed a drop of some essential oil, sometimes removes the difficulty. At others, a little coffee, orange wine, or a bitter, and occasionally slightly warming the oil, so as to render it more fluid, answer well. By these or similar methods, it is rare indeed that the oil cannot be taken.

Numerous substitutes have been proposed for cod-liver oil, such as shark, dugong, and skate oils, cocoa-nut oil, neat's-foot oil, &c. Any of these substances, including cream and butter, so long as they can be assimilated, and do not prove purgative, are beneficial in Phthisis. It will be found, however, that, of all oleaginous matters known, cod-liver oil is the most generally useful and the best. Dr. Baur, of Tubingen, recommended that it should be used externally, but extensive trial has demonstrated what physiology teaches, viz. that the skin, being only slightly pervious to substances from without, cannot be made the vehicle for introducing nutritive matter. Dr. Buist, of Aberdeen, recommended injections into the rectum, but objections to the use of constant enemata in this country are insurmountable, and although useful as a temporary measure, cannot be made available to a sufficient degree for the cure of a disease like Phthisis. What then is really required is not oil added directly to the blood, but oil digested and emulsionized by the pancreatic and other intestinal fluids; a truth which has induced Dr. Dobell to recommend that before administration it should be mixed with pancreatic juice.

In most cases where there is fever, rapid pulse, and furred tongue, cod-liver oil is no more tolerable than food. Under such circumstances it should not be insisted on. It will also be judicious, when taken for any length of time, to intermit its use now and then for a few weeks, and give in its stead a vegetable bitter. By attention to this circumstance, the medical practitioner will easily satisfy himself that in this substance he possesses a most valuable means of prolonging life, and sometimes even of causing permanent cure in

Phthisis Pulmonalis, especially when the benefits it confers are con-joined with the other methods of general treatment to be noticed.

An additional benefit has followed the obvious good effects of cod-liver oil in Phthisis, as stated by Dr. E. Smith, who says: "A prime reason of the good which has resulted from the use of the cod oil is the regular supply of fat to persons who otherwise would not have taken it in due quantity; and a great merit in the introduction of it to general use is in having led inquirers to prove the very im-portant part which fat plays in the animal system, and the real necessity for it which exists in all persons and particularly in the young."[1] This observation evidently results from the histology, pathology, and treatment of Phthisis which for so many years we have endeavoured to impress upon the profession.

Pure Atmosphere.—If it be essential for the purpose of nutrition to supply the blood with those materials which are necessary for building up the tissues and compensating the waste they undergo during their action, it is equally so that such materials should be properly prepared and fitted for the purposes to which they are to be applied. Of the various processes necessary to this end there can be little doubt that that of respiration is the chief, the object of which is constantly to introduce into the blood from the atmospheric air a certain amount of oxygen, and constantly to give off from the blood to the air a corresponding amount of carbonic acid gas. If the lungs be feeble or diseased, their action is of course diminished, a circumstance which only renders it the more necessary that no difficulty to oxygenation of the blood should be allowed to originate from a deterioriated constitution of the air itself. But this truth is one which it is exceedingly difficult to impress upon patients, the irritability of whose chests and whose susceptibility to cold induce them to close the doors and windows, and thus prevent fresh air from entering their rooms. Now, while the giving off carbonic acid gas by the lungs makes no impression upon the mass of the atmosphere at large, it soon sensibly deteriorates the amount of air inclosed in a moderate-sized room, the breathing of which is most destructive to the phthi-sical invalid. Instead of inhaling only oxygen and nitrogen, and expiring carbonic acid gas and nitrogen, they take in a sensible amount of carbonic acid at each inspiration, which poisons the arterial blood, renders it less fit for nutrition, irritates and burdens the lungs, occasions languor, bad appetite, pallor of countenance, and indeed every evil which it should be the aim of the physician to remove. Moreover, good diet and cod-liver oil must be useless unless a vigorous respiration exists at the same time, as they tend to increase the carbonaceous elements in the frame, which are mostly excreted by the lungs. A proper ventilation of the rooms occupied by the patient is therefore absolutely essential, and this rule especially applies to the sleeping room. The majority of mankind spend one-third of their life in sleep, while the invalid often remains in the bed or

[1] On Consumption, p. 348.

bedroom much longer. How important then is it to secure a pure breathing air during this period!

It is now twenty-five years ago since I became convinced of the injury of shutting up patients in their rooms during winter, and regulating the temperature, as was formerly the custom. A young man, with cavities in his lungs, who had borne confinement in this way tolerably well for a winter, found it so irksome on a second trial, that on one occasion he went out and walked to the top of Arthur's Seat. Instead of being worse, he that day ate his dinner with appetite, all his symptoms were moderated, and under the combined influence of pure air and exercise he not only was better, but ultimately worked out a perfect cure, and is now alive in good health. Since then I have had abundant opportunities of satisfying myself of the great advantages to be derived from securing free ventilation and pure air to consumptives.

These points are dwelt on as forcibly as possible, because it must be admitted that, partly as the result of custom or prejudice and partly in consequence of the severity and changeableness of the climate, a good ventilation of the house and sleeping room is, in this country, a matter of extreme difficulty. In all cases, however, it merits the especial attention of the physician. Hence he should regard the position of the house, the nature of the prevailing winds, the windows of the sitting-room, and the place in it occupied by the patient, how the bed is placed in reference to the door and windows, &c. The great end he should aim at is to surround his patients with *as much pure air as possible, consistent with warmth and absence of draughts*, a problem often very difficult to work out. There should be no curtains round the bed, an open fire should burn in the room during winter, in itself an excellent ventilator, the bed should be placed in a position free from the direct draught between the fire and the door or window, and only a moderate temperature permitted, as when in bed the patient ought not to feel cold. In summer, good ventilation should be secured by letting down the windows an inch or so at the top—an excellent method, first strongly insisted upon by Dr. McCormack, of Belfast, and one which, indeed, is at all times available in this country; unfortunately, abroad, the construction of the windows does not admit of it. The necessity of constantly breathing pure air should prevent the phthisical patient from attending crowded assemblies, *table d'hôtes*, theatres, concerts, or any amusements where the atmosphere must necessarily be deteriorated, and which, being breathed for hours, almost invariably exacerbates the symptoms and increases the malady. It is in consequence of the facility of breathing a purer air all day, and the necessary avoidance of crowded and closed rooms at night, that I am persuaded the upper classes of society experience much of the good effects of residing in certain places famed for their climate—the next point we must consider.

Climate.—It was formerly supposed that warm climates were beneficial for consumptive patients, and artificially heated temperatures,

cow-houses, and other contrivances were had resort to, to compass this end. But it will be invariably observed that unaccustomed warmth, the excessive heat of summer and autumn, or the climate of India and other tropical countries, is most injurious. Continuous frost and cold are in themselves beneficial, but by preventing the individual taking exercise in the open air they are not on that account to be recommended. What is really required is a cool temperate climate, free from great alternations of temperature, which should range from 55° to 66° Fahr. during the day, and from 45° to 55° at night. The air should be dry, or with only slight moisture, little rain, and a clear bright sun. Such an exhilarating climate, in which exercise can be taken almost daily in the open air during the winter and spring months, is the best for the consumptive patient. It exists to the greatest perfection on the north shore of the Mediterranean, between Cannes and Savona in the western, and between Spezzia and Pisa in the eastern Riviera. It may also be found in various places on the south-east coast of Spain, especially at Malaga; on the North African shore, such as Algeria and Egypt, and many other places. In the western hemisphere suitable places may be found, especially in the islands of the West Indies, and in Australia the southern shores of Victoria. The native of the British Isles who visits the sheltered nooks of the south European shore between Cannes and Pisa will be struck with the bright sun, clear atmosphere, genial yet bracing air, steady temperature, verdure, and brilliant vegetation which surround him from January to March[1]—months which at home are characterised by frost, snow, rain, fog, gloom, bleak winds, and a barren vegetation. After this period, however, the picture is reversed. Then a hot and sultry atmosphere, a scorching sun, an intolerable glare, innumerable mosquitoes, a brown and burnt-up vegetation exist, while at home there prevail a genial atmosphere, cool breezes, moderate sunbeams, a varying sky, an emerald foliage, and a charming variety of mountain and lake which gives all that can be desired. I do not know a better winter residence for the invalid than some sheltered bay in the western Riviera, where, in consequence of the sea being immediately in front of his house, and innumerable little valleys of the Alps close behind it, he can at all times protect himself from wind, from whatever quarter it may blow. Many observations have satisfied me that the still, warm, and moist relaxing atmosphere, though of the greatest service in cases of asthma, is injurious to the phthisical invalid. Great care should be taken to avoid sharp winds, and especially east winds. This at Mentone is readily done by walking out of the back door of your house directly into some protected Alpine valley. In this country, however far we go west, it is escaped with difficulty, and as a general rule

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[1] On this subject I cannot too strongly recommend the perusal of Dr. Henry Bennet's work, " Winter in the South of Europe," although the views expressed in the text are derived from personal experience, and careful examination of the great advantages referred to.

North Britain more especially should be avoided from January till the end of May. In summer and autumn, on the other hand, I am satisfied that the cool atmosphere of Scotland cannot be surpassed in benefit, especially as we find it on the shores of a Highland lake, admitting of every variety of exercise, active and passive, in the open air. Indeed, whatever advantages may result from a well-chosen winter residence, carelessness in fixing on a proper habitation during summer may more than counterbalance the good previously obtained. It is by perseverance in well-doing that the great end of cure is to be arrived at.

For winter, the best climate for the consumptive invalid in this country is the south coast, extending from Hastings on the east to Penzance on the west side, including the several stations of Bournemouth, Ventnor, Sidmouth, and Torquay. In Scotland, Rothsay, and in Ireland, Cork, are the best stations. To the large mass of persons who cannot avail themselves of even these advantages, every opportunity should be seized on of going out when the weather admits of it. It is not so much to a foreign climate itself as to the facility it affords for enjoying exercise, and free atmosphere, without the risks that prevail in Great Britain, that the benefit is to be attributed. With proper care, however, much may be done at home, and many cases have been permanently cured in this country by means of a hygienic treatment, conducted on the principles we are advocating.

For summer, the west coast of Scotland, and especially the beautiful bays on the shore of Loch Lomond, near Tarbet, offer the best residence for the consumptive. Here the immediate neighbourhood of Loch Long furnishes the visitor with all the advantages of a marine as well as of a fresh-water lake, both which are so situated that the most perfect protection from wind, combined with shade, is close at hand. It cannot be too strongly impressed upon the patient that carelessness in summer too often more than counterbalances the good results that have been obtained in winter.

Exercise.—The best stimulant for nutrition is appropriate exercise, which by accelerating the circulation and respiration, and causing natural wasting of the tissues, excites the demand for substance to repair it. It will generally be found useless to give nutriment, even when combined with pure air and good climate, unless, by means of exercise, air be forced into the lungs in somewhat increased quantity, and circulated by means of the blood throughout the system. And here it is that favoured localities are of so much value, by tempting the invalid out of the house, and permitting him to remain there, without encountering cold, wind, rain, or other risks to which he is exposed in this country. All exercise, however, should stop short of considerable fatigue. I say considerable, because some patients are always indisposed to move, and plead weakness and fatigue as incapacitating them from any exercise whatever. Walking, or riding on horseback, are the best kinds of exercise when weakness is not great. Slowly climbing a hill brings all the muscles into action, and is a good

stimulant to the respiratory and circulatory systems. All violent, sudden, and unequal exertions should be avoided. Reading or speaking aloud, singing or practising upon some wind instrument, may be permitted in moderation, when the disease is not active, but should never be long continued. As a general rule they are injurious. It is often better to take a little exercise at a time, but frequently in the course of the day, and to continue it regularly and methodically, gradually increasing its amount and varying its character as the strength improves.

For those who are weak and feel soon exhausted, passive exercise is best, such as in a carriage or in a boat, of course well wrapped up and protected from the wind. In summer, sitting or lying, well supported, in a boat pulled on a Highland lake, while, for occupation, reading, mixed with a little fishing, and the conversation of a pleasant companion—the varying tints and outlines of the landscape also serving occasionally to occupy the attention—is perhaps the most salubrious kind of exercise for the not over-weakened invalid. For the same reason long voyages at sea are beneficial. I can speak with confidence of the three months' voyage to Australia by the Cape of Good Hope, commencing about the end of October. The climate is all that could be wished for, the trade-winds assist the vessel forward, the sea breeze is invigorating, and the life on deck all that could be desired. I have known many persons, very ill on leaving, lose all their symptoms before landing at Sydney or Melbourne. Sultry heat on shore must then be carefully avoided, and the visiting neighbouring mountains or Tasmania becomes necessary in summer, in order to avoid the enervating effect of extreme heat. The return voyage should be carefully considered, and the winter at Cape Horn especially avoided.

When none of these methods are available, sitting out in the open air should always be insisted on, in a garden, on a balcony, or even at an open window, anything being better than remaining shut up in a room from morning to night.

In this, as in all other matters concerning hygiene, the patient requires to be cautioned and carefully watched. For if some feel disposed to do too little, others imagine that they cannot do too much. Under the idea that riding was beneficial, I have known a man hire a horse, and gallop about until he was so exhausted that he did not recover for a fortnight. Others in foreign hotels have taken rooms at the top of the house to obtain pure air, and not considered the excessive toil imposed upon them by having to climb the lofty stairs. Others take villas in the neighbourhood of towns, and are thus led into a daily fatiguing walk greater than their strength will sustain. Again, free exposure to the air must be conjoined with avoidance of draughts and cold winds. The rapid motion of a carriage through a dry bracing atmosphere is often too much for the invalid, who should proceed slowly. Carelessness and often an unacquaintance with these dangers are constantly producing mischief, so that the watching and regulating these matters will require all the vigilance of the practitioner.

Bathing.—There is no doubt that the relation between the skin and

lungs is very intimate, a fact better observed perhaps in Phthisis than in any other disease. When the lung can no longer exhale the large amount of watery vapour which is required, it is separated by the skin as insensible or sensible perspiration. Any sudden cold or chill affecting the skin is at once communicated to the lungs by reflex action, and excites irritation and cough. Now this susceptibility of the skin, so far from being prevented, is fostered and increased by constantly living in warm rooms, wrapping up too closely in shawls or furs, warm bathing, ointments, &c. &c. What is required is, that the skin should be kept constantly clean, and the epidermis and sebaceous matter that obstructs the orifices of the ducts daily removed by cold bathing, whereby the organ is gradually accustomed to the application of a lower temperature, and rendered less liable to be affected by changes in the atmosphere or wind. In the majority of cases also a momentary plunge into the cold bath produces a glow of heat and pleasant feeling of reaction, exciting the capillary circulation of the surface, and relieving congestion in the lungs. When, however, in consequence of weakness, such reaction is not experienced, but in its stead shivering, headache, and continued cold, then either a tepid bath should be employed, or the sitz bath, and sponging rapidly only the chest and throat should be practised. There is no better protection against catching frequent colds than daily sponging the chest with cold water. The neck and chest, however, should always be covered, the growth of beard and moustache in men encouraged, whilst women should avoid low dresses, and always be prepared with an extra shawl to throw round the shoulders, even in going from one room to another through an exposed lobby. Respirators are not useful in conveying warm air into the lungs, nature having carefully provided for this, but by acting as extra pieces of clothing, and protecting the skin of the face. An ordinary comforter, and a small shawl held in the hand to be applied to the face on encountering a sudden gust of wind, is a better contrivance.

From what has been now stated with regard to the general or hygienic treatment of Phthisis Pulmonalis it will, we trust, be apparent that all the means spoken of unite to produce one result, and that no one of them alone can be depended upon. It will be of little use giving good diet or cod-liver oil, unless a pure atmosphere enter the lungs, so that chylification may produce good sanguification, while these in their turn are directly stimulated by exercise and judicious bathing. All these operations work together for good, the object being to stimulate the whole nutritive functions, augment appetite, gradually increase the strength, arrest the onward progress of the disease, and initiate in it that retrograde process formerly described, which shall terminate in health. To arrive at this end, however, a special treatment will be required for each individual case, which we must next proceed to describe.

SPECIAL TREATMENT OF PHTHISIS PULMONALIS.—It is to the undue importance so frequently given to the special as distinguished from

the general treatment of Phthisis that the former want of success may be attributed. The management of individual symptoms and the administration of drugs, so far from being the chief, should invariably be the subordinate part of our object, and this for the obvious reason that, if nutriment succeed in checking the disease, the symptoms will disappear of themselves. At the same time it must necessarily happen in the course of every case that various symptoms and complications will press themselves upon our notice, and their palliation or removal, while still continuing our general efforts at cure, is always a matter of great importance. It is only by studying individual examples of the disease, observing the numerous and varied combinations and indications that each presents, that the difficulties the practitioner has to combat in this way can possibly be understood. I have too frequently seen patients lying in bed, ener-vated, without appetite, sweating at night, and apparently sinking, with a mass of bottles and boxes at the bedside bewildering to con-template,—each of these it is imagined has some special symptom or purpose to fulfil,—such as lozenges, drops, and mixtures, to relieve coughs; opiates and sedatives, to cause sleep and diminish irritability; catechu, gallic acid, tannin, and acetate of lead, to check diarrhœa or arrest hæmoptysis; sulphuric acid, to relieve sweating; chalk and antacids, to combat acidity and dyspepsia; quinine, iron, or bitters, as tonics; wine, to support strength; cod-liver oil, &c. &c. All these I have seen administered at intervals about the same time, so that the stomach, drenched with drugs, is utterly prevented from performing its healthy functions. Under such circumstances, suspending all such supposed remedies, or preventing the patient from having recourse to them at will, is often the best introduction to an improvement, which the cold or tepid bath, insisting on their getting up and going into the open air, has, much to their surprise, tended to increase. It follows that, in all our attempts to relieve symptoms, the utmost care should be taken not to interfere with the far more important object of arresting and ultimately curing the disease by general treatment. The various phenomena that present themselves, therefore, should be managed as follows.

Loss of Appetite and Dyspepsia.—These are the most constant and important symptoms of Phthisis, inasmuch as they interfere more than any other with the nutritive processes. If food, or its substitute cod-liver oil, cannot be taken and digested, it is in vain to hope for amelioration. Here we should avoid a mistake into which the inex-perienced are very liable to fall. Nothing is more common than for phthisical patients to tell their medical attendants that their appetite is good, and that they eat plentifully, when more careful inquiry proves that the consumption of food is altogether inadequate, and that they loathe every kind of animal diet. We should never be satisfied with general statements, but determine the kind and amount of food taken, when sufficient proof will be discovered, in the vast majority of cases, of the derangement, formerly alluded to, of the appetite and

digestive powers.   Very commonly also there will be acid and other unpleasant tastes in the mouth, loathing of food, and other dyspeptic symptoms.   In all such cases, especially if too much medicine has been already given, the stomach should be allowed to repose itself before anything be administered, even cod-liver oil.   Sweet milk, with toasted bread, and small portions of meat nicely cooked, so as to tempt the capricious appetite, should be tried.   Then ten drops of the sp. ammon. aromat., given every four hours in a wine-glassful of some bitter infusion, such as that of calumba or gentian, with a little tr. aurantii, tr. cardamomi, or other carminative.   In this way the stomach often regains its tone, food is taken better, and then cod-liver oil may be tried, first in tea-spoonful doses, cautiously increased ; or other forms of fat, such as pork fat, bacon, suet, or butter, may be tried.   Should this plan succeed, amelioration in the symptoms will be almost certainly observed.

Nausea and Vomiting.—Not unfrequently the stomach is still more deranged ; there is a feeling of nausea and even vomiting on taking food.   In the later stage of Phthisis, vomiting is also sometimes occasioned by violence of the cough, and the propagation of reflex actions, by means of the par vagum, to the stomach.   In the former case, the sickness is to be alleviated by carefully avoiding all those substances which are likely to occasion a nauseating effect, by not overloading the stomach, but allowing it to have repose.   Here also, in cases where too much medicine has been administered, a suspension of all medicaments for a few days will frequently enable the practitioner to introduce nourishment cautiously with the best effect.   I have found the following mixture very effectual in checking the vomiting in Phthisis : ℞ Naphthæ medicinalis ʒj ; tinct. cardamomi comp. ʒj; mist. camphoræ ʒvij.; M. ft. mist., of which a sixth part may be taken every four hours.   When it depends on the cough, those remedies advised for that symptom should be given.   I have tried emetics for the relief of nausea and vomiting, but with no good result.

Cough and Expectoration.—At first the cough in Phthisis is dry and hacking.   When tubercle softens or bronchitis is present, it becomes moist and more prolonged.   When excavations exist, it is hollow and reverberating.   In every case cough is a spasmodic action, occasioned by exciting the branches of the pneumogastric nerves, and causing simultaneous reflex movements in the bronchial tubes and muscles of the chest.   The expectoration following dry cough is at first scanty and muco-purulent, and afterwards copious and purulent.   When it assumes the nummular form,—that is, occurs in viscid rounded masses, swimming in a clear fluid mucus,—it is generally brought up from pulmonary excavations.   The accumulation of the sputum in the bronchial tubes is an exciter of cough ; and hence the latter symptom is often best combated by those means which diminish the amount of sputum.   When, on the other hand, the cough is dry, those remedies should be used which diminish the sensibility of the nerves.   In the first case, the amount of mucus and pus formed will materially depend on the

weakness of the body and the onward progress of the tubercle. Hence good nourishment and attention to the digestive functions are the best means of checking both the cough and the expectoration; whereas giving nauseating mixtures of ipecacuanha and squills is perhaps the worst treatment that can be employed. There is no point which experience has rendered me more certain of than that, however these symptoms may be palliated by cough and anodyne remedies, the stomach is thereby rendered intolerant of food, and the curative tendency of the disease is impeded. On the other hand, nothing is more remarkable than the spontaneous cessation of the cough and expectoration on the restoration of the digestive functions and improvement in nutrition. When the cough is dry, as may occur in the first stage, with crude tubercle, and in the last stage, with dry cavities, slight counter-irritation is the best remedy, employed in various forms. Opium may relieve, but it never cures. The occasional use of the sponge saturated in a solution of nitrate of silver is frequently of the greatest service, especially when from irritation of the fauces or larynx vomiting is occasioned.

There is a period in the history of chronic Phthisis when the cavities become dry and the sputum inspissated, tough, and difficult to expectorate. The practitioner is then frequently asked for some medicine to loosen the phlegm, relieve the feeling of tightness or compression in the chest, and dyspnœa. Under these circumstances, in no case should he resort to expectorants and opiates. The patient should be instructed that these are favourable symptoms, and indicate healing and cicatrization going on in his chest. Instead of relaxing, now is the time to persevere in avoiding palliatives which nauseate and depress the system. A few drops of sulphuric ether in camphor julep, diminishing alarm, and a little quietude, constitute all the treatment required.

Pain.—It is very surprising to what an extent tubercular disease of the lung may occasionally proceed, without causing inconvenience in the chest. Frequently there are sensations of constriction or oppression, which, however, scarcely excite attention; or from their fugitive character are attributed to any cause but the right one. Occasionally there is a fixed pain in the affected side, which is increased on coughing. This more especially occurs when there is chronic pneumonia or pleurisy. The best method of relief is to keep the parts at rest as much as possible, and apply warm fomentations or a hot poultice. Slight counter-irritation with tincture of iodine may also be tried.

On the other hand, leeches and cupping, though they may relieve, are opposed to the general principle of supporting the strength, and should be avoided. The same may be said of blisters, croton oil, tartar emetic ointment, and the moxa. I have long satisfied myself that severe counter-irritation is of no real benefit, whilst it produces an amount of suffering that irritates, and frequently does harm. Opiates are also injurious, by destroying the appetite and increasing the perspirations. At the same time, if pain be very distressing and long-

continued, and especially if it destroy sleep, some anodyne must sooner or later be had recourse to. Under these circumstances I have found chlorodyne derange the appetite, tongue, and stomach less than any other remedy of this class. Recently, chloral in fifteen or twenty-grain doses has seemed to me to act as a purer hypnotic and cause less disturbance to the economy than other remedies. Again, when all curative efforts are obviously useless, and death is approaching, palliatives need no longer be withheld. Then, all hopes of course being abandoned, relief of pain, if it exists, becomes our chief duty. But even then it should be effected with caution and discretion, otherwise the discomfort and increase of other symptoms in the patient will more than counterbalance the temporary benefit obtained.

Diarrhœa.—This is a very common symptom throughout the whole progress of Phthisis, at first depending on the excess of acidity in the alimentary canal, to which we have alluded, but in advanced cases connected with tubercular deposit and ulceration in the intestinal canal. The best method of checking this troublesome symptom is by improving the quality and amount of the food. The moment the digestive processes are renovated, this, with the other functional derangements of the alimentary canal, will disappear. Hence at an early period we should avoid large doses of opium, gallic acid, tannin, and other powerful astringents, and depend upon the mildest remedies of this class, such as chalk with aromatic confection, or an antacid, such as a few grains of carbonate of potash. When, on the other hand, in advanced Phthisis, continued diarrhœa appears, and is obstinate under such treatment, then it may be presumed that tubercular disease of the intestine is present, and the stronger astringents with opium may be given as palliatives.

Hæmoptysis.—This symptom sometimes appears suddenly, as we have seen, in individuals in whom there has been no previous suspicion of Phthisis, and in whom, on careful examination, no physical signs of the disease can be detected. On other occasions, the sputum may be more or less streaked with blood; and lastly, it may occur in the advanced stage of the disease, apparently from ulceration of a tolerably large vessel which may be dilated or aneurismal. In all these cases the best remedy is perfect quietude, and avoidance of every kind of excitement, bodily and mental. Astringents have been recommended, especially tannin, gallic acid, acetate of lead, and opium; but how these remedies can operate I am at a loss to understand; and I have never seen a case in which their administration was unequivocally useful. Can it be supposed that either of these substances can be absorbed into the blood in such quantity as to render that fluid more capable of coagulating in the lung where the vessel is ruptured? I have now met with several cases where supposed pulmonary hæmorrhage really originated in follicular disease of the pharynx or larynx, and, with the supposed phthisical symptoms, was removed by the use of the probang and nitrate of silver solution.

Sweating I regard as a symptom of weakness, and therefore as a

common, though by no means a special one in Phthisis. Here, again, the truly curative treatment will consist in renovating the nutritive processes, and adding strength to the economy. It will always be observed that, if cod-liver oil and good diet produce their beneficial effect, the sweating, together with the cough and expectoration, ceases. On the other hand, giving acid drops to relieve these symptoms, as is the common practice, by adding to the already acid state of the alimentary canal, is directly opposed to the digestion of the fatty principles, which require assimilation.

It should not be forgotten that consumptive patients, and all those suffering from pulmonary diseases, are especially sensitive to cold. The impeded transpiration from the lungs in such cases is counter-balanced by increased action of the skin, which becomes unusually liable to the influence of diminished temperature. Again, cold applied to the surface immediately produces, by reflex action, spasmodic cough and excitation of the lungs. Every observant person must have noticed how cough is induced by crossing a lobby, going out into the open air, a draught of wind entering the room, getting into a cold bed, &c. &c. The mere exposure of the face to the air on a cold day takes away the breath, induces cough, and obliges the patient in-stinctively to muffle up the mouth. The numerous precautions, therefore, that ought to be taken by the phthisical individual, should be pointed out, especially the necessity of warm clothing, to which large additions should be made on going out into the air. Thus, covering the lower part of the face is important as a means of extra clothing, and not as a means of breathing warm air, as the favourers of respirators imagine. The patient should always sit with his back to the horses or to a steam-engine, and if by accident his shoes or clothes become wet, they should be changed as soon as possible. In the house, ladies should have a shawl near them, to put on in going from one room to another, in descending a stair to dinner, &c. By attention to these minutiæ, much suffering and cough may be avoided.

Febrile Symptoms.—The quick pulse, general excitement, loss of appetite, and thirst, which are so common in the progress of phthisical cases, are dependent on the same causes as those which induce symp-tomatic fever in general. Vascular distension, resulting in exudation and its absorption, is proceeding with greater or less intensity in the lungs, and frequently in other organs. This leads to nervous irritation and increase of fibrin in the blood, accompanied by febrile phenomena. The intensity of these is always in proportion to the activity of local disease, or to the amount of secondary absorption going on from the tissues, or from morbid deposits. Nothing is more common than attacks of so-called local inflammations in Phthisis, and the careful physician may often determine by physical signs the supervention of pleurisy, pneumonia, or bronchitis on the previously observed lesion, and not unfrequently laryngitis, enteritis, or other disorders. In such cases, nature herself dictates that the analeptic treatment, otherwise appropriate, is no longer applicable—food disgusts, and fluids are

eagerly demanded. Under these circumstances, it has been common to apply leeches to the inflamed part, and extract blood by cupping, measures which undoubtedly cause temporary relief, but which are wholly opposed to the plan of general treatment formerly recommended, and to what we know of the pathology of the disease. Every attack of febrile excitement is followed by a corresponding collapse, and it should never be forgotten that, in a disease which is essentially one of weakness, the patient's strength should be husbanded as much as possible. Hence the treatment I depend on in such circumstances consists of at first the internal administration of the neutral salts, combined with diuretics, in order to favour crisis by the urine. Subsequently quinine is undoubtedly advantageous. I have satisfied myself that such attacks are not to be cut short by leeches or cupping, and although in many cases, as previously stated, temporary relief is produced, the exposure of the person, and unpleasant character of the applications, the trickling of blood, and wet sponges, as often irritate, and give rise to unnecessary risk. Still there are cases where topical blood-letting, if it cannot be shown to have advanced the cure, cannot be proved to have done harm ; but these cases, as far as my observation goes, are very few in number. In the rapidly febrile cases, or the so-called instances of acute Phthisis, mercury has been recommended, but has never produced the slightest benefit.

Debility.—This is a very common symptom of Phthisis from the first, and frequently leads the patient into indolence both of mind and body, a condition very unfavourable for the nutritive functions, upon the successful accomplishment of which its removal depends. It is to remove the weakness that tonics have been administered, but I have never seen quinine, bitter infusions, or even chalybeates, of much service alone, while the continual use of nauseous medicine disgusts the patient, and interferes with the functions of the stomach. Neither have I ever been able to satisfy myself that the hypophosphites of soda or of lime, or the syrup of those phosphates and iron, have ever been of service. In all cases, the removal of debility is to be accomplished by counteracting the dyspeptic symptoms, giving cod-liver oil, an animal diet, and improving the appetite by gentle exercise and change of scene. Should the practitioner succeed in renovating the nutritive functions, it is often surprising how the strength increases, in itself a sufficient proof as to what ought to be the method of removing the debility. I have frequently seen patients who have been so weak that they could not sit up in bed without assistance so strengthened by the analeptic treatment, that they have subsequently walked about and taken horse exercise without fatigue, and this after all the vegetable, mineral, and acid tonics have been tried in vain.

Despondency and Anxiety.—It is impossible for the careful practitioner to avoid noticing the injurious influence of depressing mental emotions on the progress of Phthisis. Indeed the worst cases are those of individuals with mild, placid, and unimpassioned characters, who give way to the feelings of languor and debility which oppress them.

Such persons are most amiable patients—they give no trouble—anything will do for them—they resign themselves to circumstances, and state that they are eating well and getting better up to the last. These are cases of bad augury, for it is exceedingly difficult to inspire them with sufficient energy to take exercise, or to carry out those regulations which are absolutely essential to renovate the appetite and the nutritive functions. Such persons are benefited by slow travelling, cheerful society, and everything that can elevate the spirits, and, insensibly to themselves, communicate a stimulant to the mental and bodily powers. Anxiety, on the other hand, though it may sometimes depress and interfere with the digestive functions, is often a most useful adjunct to the physician. Those who experience it are most careful of their health, sometimes indeed too much so; but, if once satisfied of the benefit of any particular line of treatment, they pursue it with energy. These are cases of good augury, and most of the permanent cures I have witnessed have been in such persons—medical men, and others acquainted with the nature of their disease, who have exhibited resolution and a noble fortitude, who have bravely struggled against local pain, general debility, and nervous fear, and literally fought the battle of life with the greatest success.

When the disease has been arrested, all the symptoms have disappeared, and even some degree of *embonpoint* returned, the patient must still be careful, still consider himself an invalid, and continue to pursue the hygienic regulations which have proved so beneficial. These, however, will not materially interfere with his enjoyment of life, or even the pursuit of active business or professional life. Amongst the poorer classes, it will be more difficult to obtain such handiwork or occupation as may not be injurious. In order to live, however, they must exchange their unhealthy for more healthy modes of life. As a general rule, the dwellers in towns should seek the country, and the inhabitants of rural districts change the scene of their labours—always remembering that it is not mere place that can benefit, but the opportunities it may offer for carrying out that improvement in the nutritive functions we have endeavoured to show is so necessary.

Local Treatment.—It has not failed to suggest itself to medical practitioners that remedies might be useful if applied directly to the lungs. To this end condensed air, an oxygenated atmosphere, carbonic acid, sulphurous and tar fumes, and all kinds of substances in a gaseous form have been inhaled. Solutions in a state of vapour, or divided into spray, have also been tried. Astringent and other fluids have been injected down the larynx and bronchi. Pulmonary cavities have even been opened from without, and variously treated with a view of causing cicatrization. The result of all these efforts has been —what an intelligent consideration of the pathology of the disease might have anticipated—a uniform failure.

STATISTICS.—It is a matter of extreme difficulty to determine with exactitude how the change in the treatment of Phthisis which commenced in 1841, and became pretty general in 1850, has influenced

he mortality of Phthisis Pulmonalis. In 1852, Dr. Wood, of Philadelphia, remarks of it, that in that city, during the ten years from 1840 to 1849 inclusive, the average proportion of mortality from Phthisis was 1 in about 6·76 from all causes, or 14·8 per cent., and the same average existed in previous years. Cod-liver oil was then generally used in its treatment, and the mortality sank in this disease during 1850-51 to 1 in 8·33, or about 12 per cent., and in 1851 it was only 11·86 per cent.

In 1862, Dr. C. J. B. Williams, in one of the Lumleian lectures delivered to the London College of Physicians, observes that the experience of Louis and Laennec gave an average duration of two years' life in Phthisis after it was decidedly developed, but that, since cod-liver oil was introduced, he infers from 7,000 cases that the average duration of life has been four years.

The registration of deaths in Scotland only commenced in 1855, and offers therefore no means of comparison, as regards Phthisis Pulmonalis, between the mortality occurring before and after that period. But the English registration of deaths commenced in 1837, and, with the exception of a few years, has continued up to the present time. The following is the result :—

Years.	Average annual population.	Average of total number of deaths.	Average of deaths from Phthisis.	Percentage of deaths from Phthisis to total deaths.
37–41	15,720,385	347,070	55,718	16·0
50–54	18,174,011	359,681	50,515	14·0
55–59	19,257,184	425,703	50,187	11·3
60–64	20,196,787	495,531	51,595	10·4

It would appear from the above table that, taking a five years' average previous to 1841, before cod-liver oil and an analeptic treatment were introduced, the proportion of deaths from Phthisis was 16 per cent.; whereas, in the years 1850 to 1854 inclusive, the deaths were 14; in 1855 to 1859, 11·3; and in 1860 to 1864, only 10·4 per cent. of the deaths from all causes. It must be observed, however, that a certain number of cases annually are vaguely returned as "lung diseases," and that whilst deaths from Phthisis have diminished, those from pneumonia and bronchitis have greatly increased. Doubtless exactitude in diagnosis has very much extended among medical practitioners during the last twenty years, whilst it is a matter of common observation that the winter and spring seasons have increased in severity and duration, circumstances which to a certain extent might account for the numerous returns of pneumonia and bronchitis. Without attaching, therefore, too much importance to the exactitude of the results obtained by the Registrar-General, all that can be said is, that as far as they can be relied on, they exhibit during the last twenty-five years a marked diminution in the mortality of Phthisis Pulmonalis, as compared with the period before cod-liver oil and a restorative treatment were employed.

CANCER OF THE LUNGS.

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Literature.—*Hollerius*, Op. omnia, De Morb. intern. 1674; *Heister*, De Asthm. schirr. 1749 ; *De Haen*, Ratio medend. Parts v., vi. 1765 ; *Morgagni*, Epist. i. xxii. art. 22; *Ib.* Epist. xx. art. 39, 1780 ; *Van Swieten*, Comment. ad Aptor. Part ii. p. 797; *Bayle*, Recherches sur la Phthisie pulmon. Paris, 1810 ; *Langstaff* and *Lawrence*, Med. Chir. Transact. viii. p. 272; *Langstaff*, Med. Chirurg. Transact. ix. p. 297; *Andral*, Clinique Médicale, 1830 ; *Cailliot*, Sur l'Encéphaloïde, 1833 ; *Williams*, Diagnosis of Diseases of the Chest, 1835 ; *Durand-Fardel*, Journal Hebdomad. 1836 ; *Laennec*, Traité de l'Auscultation, 1837 ; *Stokes*, Diseases of the Chest, 1837 ; *Strave*, De Fungo pulmonal. 1839 ; *Kleffaus*, De Cancr. pulmon. Gröning, 1841 ; *Marshall-Hughes*, Guy's Hosp. Rep. 1841 ; *Watson*, Lond. Med. Gazette, 1841 ; *John Simon*, General Pathology, 1850 ; *Lebert*, Traité des Maladies cancéreuses, 1861, and his Anat. Pathol. 1855—1862; *Ebermann*, De Cancro pulmon., Petropolis, 1857 ; *Bright*, Guy's Hosp. Rep. v. p. 377 ; *Harrison*, Dub. Journ. xvii. p. 326 ; *Green*, Dub. Journ. xxiv. p. 282 ; *Tiniswood*, Monthly Journal, July 1844 ; *Burrows*, Med.-Chir. Trans. xxvii.; *Maclachlan*, Lond. Med. Gaz. 1843 ; *King*, Ibid.; *Köhler*, Krebskrankheiten, 1857 ; *Pemberton*, On Melanosis, Midland Quarterly Journ. of Med. Science, May 1857, p. 129 ; *Bright*, Diseases of the Heart, Lungs, &c. 1860 ; *Aviolet*, Du Cancer du Poumon, Paris, 1861 ; *Begbie*, Archives of Medicine, 1861 ; *Rokitansky*, Pathol. Anat. 1861, vol. iii.; *Walshe*, Diseases of the Lungs—on Cancer, 1863 ; *Skrzeczka* in *Virchow's* Archiv, vol. xi. p. 179 ; *Virchow's* Geschwülste; *Cockle*, On Intrathoracic Cancer, 1865; *Andrew*, Primary Cancer of the Lungs, Transact. Path. Soc. 1865, p. 51; *Charles Moor*, Report on Cases of Cancer, Brit. Med. Journ. 1866, vol. ii. ; *Rindfleisch's* Pathologische Gewebelehre, Dritte Lieferung, 1868.

Cancer of the Lungs is by no means a frequent occurrence. Bayle observed only three cases at the post-mortem examination of 150 individuals who died of phthisis. Begin, at 200 dissections, has only twice observed the disease. Herrich and Popp found malignant growths in 68 out of 1,171 corpses ; but amongst these 68 there were only six cases of Cancer in the Lungs. Recent observations by Dr. James Russell, Dr. Andrew, and others, have, however, confirmed

the opinion held by excellent observers, that the lungs may not only be the only affected organ, but in secondary cancer be really a place of predilection.

Walshe considers " Cancer in the Lungs to be particularly common as the secondary development, where the testicle has been the primary seat of the disease;" whilst Dr. Day, of Stafford, appears strongly inclined to consider it more frequently a sequence of cancer of bones than of any other primary cancerous development.[1]

The truth is that cancerous affection of the lungs is comparatively common after primary development, both in the testicles and bones, but that other organs may also—though not with equal frequency—be the nidus for primary deposits, which then may be followed by secondary Cancer in the Lungs. But it must be borne in mind, that the place of primary deposits sometimes is revealed only at the post-mortem examination, which fact leads us to believe that many cases, recorded as primary Cancer in the Lungs, have been in fact secondary affections, and that the organ in which primary deposits have been formed was overlooked.

Concerning the *age* which seems most liable to be attacked, we learn from Ebermann that in 72 cases the following relations are recorded :—

From	1 to	9 years,	1 individual.
,,	9 ,,	19	,,	1	,,
,,	19 ,,	69	,,	66	,,
,,	69 ,,	79	,,	3	,,
,,	79 ,,	89	,,	1	,,

It appears, then, from this table, that the disease is rare before the age of 20, when it becomes frequent during a long period. It may be mentioned that of 78 cases in which the *sex* had been noted, 51 occurred in men; so that the ratio, therefore, was eight to three.

Concerning the forms in which Cancer of the Lungs may be observed, *colloid* is extremely rare, *scirrhous* very rare, but *encephaloid* comparatively common. In fact, some first-rate observers—Bayle, Laennec, and others—consider encephaloid the only species of cancer to be found in the lungs. This form, likewise called *medullary carcinoma*, which has received its name from the striking resemblance to brain, being thus the prevalent form of Cancer in the Lungs, to which the whole clinical interest is attached, it seems but right that, in a work like this on practical medicine, our remarks on Cancer of the Lungs should principally be confined to that form.

PATHOLOGICAL ANATOMY.—Encephaloid, as already mentioned, so much resembles the medullary substance of the brain, that, for the unaided eye, it would sometimes be difficult to say whether it be brain or pathological growth. Its consistence is generally soft, pulpy, and depends upon the amount of stroma present, the meshes of which contain the creamy fluid, generally known as *cancer juice*. The vessels

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[1] Med. Times, 1866, vol. ii. p. 230.

traversing the fungus have but thin walls, which sometimes rupture, and, admixing blood and clot with the medullary matter, give rise to the modification of encephaloid, which has been called *Fungus hæmatodes.*

In the early stages of development it is not the extravasation of blood which tinges the growth, but the abundance of very minute vessels traversing the growth, and detectable only by the aid of the microscope. Their walls are very thin and transparent, and easily liable to break. The extravasation extends through the cancerous mass in the same way as it does through the tissue in apoplectic effusions, and the pleural cavity sometimes also contains a clot of pure blood.

If, on the other hand, the cancer-cells contain black colouring matter—probably a modification of the colouring matter of the blood —the growth, of course, assumes a dark appearance, and is then called " Cancer, or Fungus Melanodes." According to Rokitansky, this species is observed only in cases of general cancerous cachexy, or, in other words, as a secondary form ; but Dr. Rogers mentions that it appears also as a primary affection.

Of 60 cases of melanosis collected and published by Pemberton,[1] the post-mortem appearances were recorded in only 35. Of these 35 cases, 17 exhibited deposits in the lungs ; but there is no practical difference between encephaloid, fungus hæmatodes, and cancer melanodes.

Secondary Cancer of the Lungs is rarely limited to these organs, but generally involves the adjoining parts, as costal pleura, pericardium, heart, diaphragm, bronchi, vessels, and nerves ; or the Cancer may, on the contrary, take its rise in one of these organs, and during its progress involve the lungs.

The bronchi may become compressed or filled with cancerous matter and their walls corroded. The arteries, but not the veins, enjoy a certain immunity when traversing a cancerous growth. The glands generally participate in the infiltration and transformation of structure ; the mediastinal glands particularly may grow into an enormous and highly vascular, cerebriform mass of several (seven) pounds weight,[2] traversed by the aorta and pulmonary artery, which may become compressed, and even converted into a very thin, soft, yellow elastic band.

I have met with an extremely rare case; the patient was a woman, aged 59 years. She was several times operated on for Cancer in the right breast, but the growth always recurred. Ultimately the lungs became involved and the patient died. At the post-mortem examination, large encephaloid masses were found at the root of the left lung, and both lungs were infiltrated with medullary cancer But the mediastinal and a very great number of bronchial glands had been changed into large dark-coloured lumps of Cancer melanodes.

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[1] Midland Quarterly Journal, May 1857, p. 145.
[2] A case under the care of Dr. Rees : Lancet, 27th August, 1864. See also Dr. Fr. Braun's " Das Vorkommen des Williamsche Tracheal Tones ;" Erlangen, 1861.

Of the nerves, by their anatomical relations, the vagus and recurrentes are particularly liable to become involved in the process, and to be materially altered. Amongst the cases contained in Dr. Cockle's most elaborate and able work on Intrathoracic Tumours, the one simulating laryngeal phthisis is of particular interest, in which "the cervical portion of the left par vagum was manifestly enlarged."[1] The shape in which the heteroplastic growth under our consideration may be found, varies very much from numberless miliary dots to cancerous tumours of twelve or fourteen pounds weight. In other instances, the lung may preserve its shape, but its normal tissue be entirely destroyed, or rather replaced by cancerous matter. In other instances again, cancerous patches may be observed with intermediate healthy tissue, or, which is the most common, the different forms co-exist—miliary deposits in one spot, nodules or nodes and larger growths in another, while a third part may be infiltrated.

As an extremely rare occurrence, which has been observed only a few times, is the form which Rokitansky has called *cancerous pneumonia*, and in which the tissue of the lung may be compressed but otherwise normal, whilst the air-cells are filled with detritus, fat globules, and principally with cancer-cells. Such a case has recently been published by Dr. Skrzeczka.[2]

The diseased lung is generally adherent to the inner surface of the sternum and ribs, or it may be compressed or retracted, entirely uncovering the heart, and most closely agglutinated to every part and organ contiguous to it. In cases of compression of one lung, the other generally becomes dilated, in order to compensate for the diminished size of the diseased one.

In some instances, the cancerous formations are limited to the costal or pulmonary pleura ; and often assuming a shape which has been compared to "wax-drops,"—Cruveilhier's "Plaques squirrheuses" —do not penetrate into the lung-tissue or air-cells, but remain superficial. In other instances, nodular deposits are formed in the very substance of the lung, growing in a centrifugal direction, and breaking through the pleura.

If cancerous derangements of other organs than the lungs have proved the immediate cause of death, but few—four or five—cancerous spots, of the size of a pea only, may be found in the lungs.[3]

The tumours, of course, undergo the same changes as cancer generally does. The softening begins in the centre, and, advancing towards the periphery, gives rise either to cancerous ulcers or, which is a rarer occurrence, to a cavern filled with puriform, bloody, and putrid juice ; the walls of such a cavity are generally thick, infiltrated with its contents, and are likewise in a state of disintegration.

On microscopic examination, the encephaloid is seen to consist of two distinct formations, the one being the *stroma*, forming differently

[1] Dr. Cockle, On Intrathoracic Tumours, vol. ii. p. 109.
[2] Virchow's Archiv, vol. xi. p. 179.
[3] Garrod, in the Lancet of 1867, vol. i.

shaped and sized meshes, which consist of fibrous bundles, partly or totally converted into an agglomeration of fatty molecules.

The consistence of the encephaloid depends upon the density of the stroma.

From these meshes the other formation, viz. the so-called *cancer-uice*, can easily be squeezed, and appears as a creamy semi-liquid fluid. The microscope reveals its colour, as depending on an abundant amount of spindle-shaped and other cells, which contain one or more large nuclei and blastema.

The cells are generally in a state of retrograde formation, or fatty degeneration, which causes their contours to appear more distinct. In a still more advanced stage, the cells become completely transformed into an agglomeration of fatty molecules.

SYMPTOMS.—Not unfrequently the patient exhibits but slight symptoms, if any, even when the disease is already far advanced. This is particularly the case with secondary, less frequently in primary Cancer, and depends on the nodular formation of the disease; for these nodules being surrounded by normal lung-tissue, permeable to the air, render auscultation and percussion useless. Dr. Stokes relates a remarkable case, illustrating not only the comparative slightness of symptoms, but also the rapidity of growth. The patient was under the care of Dr. Little, in Sligo Infirmary.[1] A young man was brought in, simply dying from a diseased leg which had been neglected. Dr. Little conceived that the only possible means to save life was amputation above the knee, which he did with the happiest result. Hectic fever disappeared, and in four or five weeks the patient had increased a stone and a half in weight; but he came back shortly, complaining of pulmonary irritation, and died in a fortnight after re-admission, when it was found that both lungs were converted completely into cancerous masses. The rapidity of growth in isolated cancerous masses was very singular. Yet, in the majority of cases, there exist symptoms enough for the formation of a strict diagnosis. The symptoms generally met with may be arranged in the following manner:—

1. *General appearance of the patient.*—Cases which run through *all stages* without apparent alteration of the patient's general health, are exceptional. Generally, the health is impaired in one or another way; and if there exists anything in disease which may be called "the habit" of that disease, I should be inclined to speak of a "cancerous habit." It may perhaps be difficult, nay impossible, to describe appropriately this habit, but a practitioner's eye trained to observe diseases and to notice even slight alterations in the countenance of his patients will surely discover it.

There is something inexpressibly painful and anxious in the lineaments of patients labouring under cancerous affections, which is not met with in any other disease. Nor is the characteristic tint of

[1] Medical Times, Sept. 1, 1866.

the patient's skin often absent. Rapidly progressing emaciation is another concomitant of Cancer; and fever of hectic nature, a rapid, small, irregular pulse, which throbs 100 to 130 times in a minute, generally are present to the last moment of the patient's life.

The literature of the disease under consideration furnishes us with numerous cases, the course of which has been precisely similar to that of phthisis; colliquative night-sweats, diarrhœa, exacerbating fever, copious expectoration. In such cases, errors in diagnosis are not only excusable, but unavoidable, and such errors have been committed. The appetite is likewise mostly deficient; the natural functions in disorder; and sleep, either by pain, dyspnœa or other causes, interrupted or entirely disturbed, and languor and debility, take possession of the poor patient.

2. *Shape of the thorax.*—The thorax may become altered in two directions, being either increased or diminished in bulk. In both instances the alteration may extend over the whole diseased side, or be partial.

Enlargement of the thorax will be observed, when, by heteroplastic growth or effusion into the pleural cavity, pressure is exercised from within upon the chest-walls; whilst diminution of the volume of the thorax will ensue from decrease of the organs situated within the chest, thus allowing the atmospheric pressure and certain muscles to act from without upon the walls of the thorax, in such a manner as to cause loss of its curved shape, and to produce flattening and depression at certain points.

The same effect may be brought about by adhesion of the pulmonary to the costal pleura. The alteration may sometimes occasion a difference between the one side of the thorax and the other, amounting to six or eight inches.

In other instances, the alteration is but slight and discernible rather by inspection than by measurement.

The movement of the thorax during respiration, depending in a very great measure upon the permeability of the lungs to air, will alter under the same conditions as if the lungs had undergone infiltration by other diseases, or had been compressed by fluid or air in the pleural cavity.

3. *Auscultation and Percussion.*—It need scarcely be mentioned that the physical signs will correspond with, and depend on, the state of the organs contained in the chest. We are aware from the principles of physical examination, that separate cancerous nodules, though they may exist in a very great number, do not exercise any influence upon the normal respiratory sound, nor do they materially alter the sound on percussion. The tissues surrounding the cancerous nodules, lose their contractility, and would give a tympanitic sound, if its tympanic character were not injured by the solid nature of the newly-formed nodules.

When the nodules become confluent, and the deposits are large, they of course interfere with normal respiration; and, according to

their nature and extension, the normal sounds of auscultation and percussion will be altered.

4. *Cough.*—Cough may exist and continue in a slight degree, so as to deceive in respect to the real nature of the disease, both the patient and the physician. But the cough may increase, and become so violent as to resemble hooping-cough, and to torment the sufferer day and night. If the disease be confined to one lung, or if one pleural cavity become filled by effused fluid, cough and shortness of breath set in from very evident causes, as soon as the patient tries to lie on the healthy side. Implication of the one or both vagi in the cancerous process will, of necessity, also be followed by frequent distressing cough of a laryngeal character.[1]

5. *Expectoration.*—It is in some cases entirely absent, but in others very copious, muco-purulent, separating into two or more layers when allowed to stand undisturbed in a glass or any other appropriate vessel. The lowest layers frequently containing so-called cancer-cells, or masses of Cancer, afford conclusive assistance in forming a diagnosis.

When a communication exists between a broken bronchus and cavity, and disintegration is going on, the expectorated matter is sometimes unbearably fœtid, and contains elastic fibres and detritus of lung-tissue. In case of corrosion of a vessel, hæmoptysis sets in, and may possibly immediately endanger life. Admixture of small quantities of blood with the sputa is neither a rare occurrence, nor of great importance.

In the above-mentioned case of communication between a bronchus and a cavern, large cancerous masses, with an admixture of blood, may be expectorated, as has been observed by Andral, Bayle, Hartman, Langstaff, or the sputa consist only of blood, and the expectorated masses are of a dark brownish colour, as described by Stokes, Burrows, and others.

6. *Pain.*—The lancinating pain, which forms a most distressing symptom of cancer in other parts of the body, is happily a comparatively rare occurrence in Cancer of the Lungs. When present, it is by no means restricted to the diseased organs, but extends to parts distant from the original place of affection. This is easily explicable by the anatomical distribution of the nerves, on which pressure may be exercised, or by the compression, embolism, or thrombosis of large blood-vessels, which may prevent proper circulation in distant parts, and even cause gangrene.

I have observed a very interesting case in a female fifty-two years of age. She had been operated on for Cancer in the left breast. Three years after operation she was suddenly seized with violent pains in the chest, lasting for some hours, disappearing then, and reappearing several days. The pain was so excruciating, that the patient in one of the paroxysms attempted suicide, but was prevented from committing it. When she was free from pain, she had neither cough nor

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[1] Cockle, loc. cit. vol. ii. p. 106.

any other sign of chest-disease. Her previous history, together with her present state, confirmed my opinion on the case as being one of intra-thoracic cancer. About a fortnight before her death, which occurred six months after I had first examined her, she began to cough and to waste away with remarkable rapidity ; and three days before death the left lower extremity exhibited symptoms which left no doubt that circulation had ceased in it. At the post-mortem examination, both lungs were found studded with small cancerous tumours, the largest of the size of a pea, leaving between them healthy tissue. The root of the right lung was involved in a large cancerous soft mass ; the liver likewise contained a considerable number of cancer-nodules, and the left iliac artery was entirely closed by a firm thrombus.

7. *Dyspnœa and Palpitation of the Heart.*—Dyspnœa may exist in a very troublesome degree even when the physical signs are still insig-nificant ; such will particularly be the case when the lungs are filled with miliary deposits. But the same may take place, the lung being but little or not at all affected, when pressure is exercised on those vagus-fibres which are inserted into the lungs. Physiology teaches that such pressure will cause acceleration of the respiratory movements, whilst irritation of those branches of the vagi, which reach the upper part of the larynx, retard these movements. In both instances, dyspnœa may be the result, and this again may become the cause of palpitations. These, however, are generally the consequence of the implication of the heart or pericardium in the disease, be it indi-rectly by pressure, displacement, &c., or by direct participation in the cancerous depositions.

Displacement of the heart by tumours or fluids will, of necessity, alter the action of the heart, which, according to Louis, is smaller in persons dying of Cancer than of any other disease. In such cases it seems to waste in common with the other tissues of the body, and becomes still more contracted from the quantity of the circulating fluid being so much diminished.

It needs no explanation to prove that degeneration of, or infiltration into the lungs, compression or closure of the larger bronchi, their being filled with cancerous matter, or the effusion of fluid into the pleural cavity, will likewise be followed by dyspnœa, or—particularly at more advanced stages of the disease—by orthopnœa.

8. *Dysphagia* is oftener connected with intrathoracic tumours of considerable size than with Cancer of the Lungs. It is always the result of pressure on the œsophagus, or of swelling of that organ in consequence of pressure. In very rare cases dysphagia may exist as a reflex action, but then it will exhibit a remittent character, whilst it will remain stationary when dependent on pressure ; in some cases the symptoms will appear as soon as the patient assumes a certain position, wherein the tumour is allowed to exercise pressure upon the œsophagus. Dr. Cockle's work contains cases illustrating both kinds of dysphagia.[1] This symptom may exist in so high a degree, and the

[1] Cockle, loc. cit. vol. ii. pp. 107, 144.

compression of the œsophagus may be so complete, as not even to allow fluids to pass, and it may become necessary to feed the patient by nutrient injections.

9. *The Voice* of a patient suffering from Cancer of the Lung is liable to many alterations. A deep bass may become altered into a high treble, or into hoarseness, according to the different causes, viz. pressure on the recurrent nerves, compression of the trachea or direct affection of the larynx by the disease. In more advanced stages of Cancer of the Lung, as well as of tuberculosis, there is scarcely a case in which the voice would not be altered in some way. According to Dr. Cockle, extinction of voice may exist without any sign of obstruction in the larynx, and without either stridor or dyspnœa, being dependent solely on paralysis of the laryngeal muscles, consequent on pressure upon the nerves by the cancerous mass within the chest. By means of the laryngoscope, such an affection in our days will be recognised during the patient's life. In a case of complete aphonia, it was observed by Andral at the post-mortem examination, that a cancerous mass had been exercising pressure on the inferior laryngeal nerves.

10. *Contraction of one or both pupils* as a symptom of intrathoracic tumour, and as due to interference with the sympathetic nerve, was first pointed out by Dr. Gairdner. Though this symptom is not pathognomonic, viz. characteristic either of Cancer in the Lungs, or of intrathoracic tumour, yet its presence may, in some instances, form a valuable link in the chain of symptomatic evidence.

11. *Effusion into one or both pleural cavities* is another symptom which is comparatively more often met with in cases of intrathoracic cancer than of Cancer of the Lungs. If present, the lung is often adherent to the vertebra, drowned as it were in the fluid, and compressed sometimes to the size of a fist, but may otherwise remain healthy in structure. If the lung-tissue, under these circumstances, is in an infiltrated state, we have a remarkable instance of an organ being infiltrated with a new formation and, at the same time, diminished in size.

The effused fluid has generally a limpid, yellow appearance, and contains albumen. The effusion generally takes place with great rapidity, and when paracentesis has been performed it is replaced in the same manner.

A case published by Dr. Begbie, in the " Archives of Medicine," in 1861, is of great interest in respect to the symptoms under consideration. The patient was a quarryman, 50 years of age, who came to the Edinburgh Royal Infirmary, desirous of obtaining advice for what he thought a slight affection of the chest. The symptoms had become troublesome only ten days before Dr. Begbie saw the patient, who, on being obliged to leave off work, had consulted a medical man in his neighbourhood. This gentleman ordered some cough-mixture, and applied a mustard-plaster over the chest; but the symptoms became worse. When Dr. Begbie saw the patient, he diagnosed intrathoracic

cancer, and, from the 24th of September to the 16th of October, 550 ounces of fluid were drawn from the enlarged chest. The patient eventually died, and primary mediastinal and pulmonary cancer was found at the post-mortem examination.

It must be borne in mind that cancerous infiltration into the lungs may progress so rapidly as to be mistaken for effusion into the pleural cavity. Mr. Middleton brought such a case under the notice of the Pathological Society of London, at the meeting on the 14th of November, 1850. During life, several medical men concurred in the opinion that the phenomena which the patient exhibited could only be due to effusion into the right pleural cavity. But at the post-mortem examination it was found that very rapid infiltration, and enlargement of the right lung, had taken place. Such cases we must bear in mind, in order to examine thoroughly and very carefully before we decide on performing the operation of paracentesis.

12. *Fever* is generally moderate, of hectic type; the pulse but little accelerated; the aid of the thermometer is, however, of great importance, for though the temperature may be normal, or but little raised, the daily exacerbation will not escape attentive observation. The pulse increases likewise towards evening, and each exacerbation is followed by perspiration, which in many cases is, indeed, very profuse and quite as violent as that which occurs in phthisis, and exhausts the patient in an extreme degree.

DIAGNOSIS.—Primary Cancer of the Lungs, in the majority of cases, admits of no diagnosis. Physical examination tells us whether or not alteration of the lung-tissues has taken place, whether or not the pleural cavity be filled with fluid or solid; but we remain ignorant of the nature of that alteration. In rare cases only, a suspicion will arise; but, unfortunately, the post-mortem examination will finally show whether our opinion has been justified, or based on wrong conclusions. Microscopical examination of the sputa should never be neglected, it being one of the principal means by which the real nature of the disease may sometimes be revealed. "I have seen many instances," says Dr. Williams,[1] "and others are on record, of ulcerous cavities formed in melanose and encephaloid solidifications of the lungs, and the expectoration in one case of a black and red, and in the other of a streaky, whitish, sanguinolent, and puriliginous matter, led to a suspicion of the nature of the disease before death."

The diagnosis of secondary Cancer generally does not afford such insurmountable difficulties as many believe. Its appearance, after primary deposits have been made in, and eventually removed from, other organs, will very often serve as a guide for our conclusion. In fact, if after the removal of a malignant growth, pulmonary or bronchial symptoms of any kind appear, it is but wise to suspect them as the beginning of the occurrence of Cancer; at all events let us be on our guard, and not treat these symptoms as if they would occur

[1] Pathology and Diagnosis of Diseases of the Chest, p. 154; London, 1835.

in persons in whom no signs of cancerous diathesis have ever made their appearance.

It is in these cases in which Hutchinson's much-neglected instrument, the spirometer, will afford good services. Individuals from whom Cancer of any organ has been removed, should, after operation, from time to time be measured in respect to the capacity of their lungs. If the amount of air evidently becomes diminished, gradually or suddenly, then we shall seldom be wrong in assuming that cancerous deposits have been made, and respectively are still progressing.

But, notwithstanding our greatest care and attention, we shall meet —and that not seldom—with cases in which a strict diagnosis will either prove impossible, or be made only after repeated examination and closely watching the case for a longer period. The diseases which are particularly liable to be confounded with Cancer of the Lungs are *chronic pleurisy with effusion into the pleural cavity, tubercular infiltration,* and *aneurism.*

DIFFERENTIAL DIAGNOSIS.—1. *Chronic Pleurisy with effusion into the pleural cavity.*—Though the consistence of encephaloid may be of a semi-fluid nature, yet it will differ in many points from effusion in respect to the symptoms as revealed on physical examination. The area of dulness on percussion, in different positions of the patient, never so strictly follows the laws of gravity as in cases of effusion. Another point of importance is, that in chronic pleurisy the area of dulness sometimes diminishes, which is particularly the case after much perspiration, or after exhibition of diuretics, or similar medicine; but the Cancer, once formed, will under no circumstances decrease.

It is true that, as Dr. Cockle says, "In many cases, mere physical diagnosis is utterly incompetent to decide the question, inasmuch as chronic pleurisy constitutes in itself an integral part of the natural history of intrathoracic cancer." But in this instance, viz. when during the cancerous process effusion into the pleural cavity has taken place, we have not any more to decide between Cancer and pleurisy with effusion; it is evident that physical examination has contributed its share towards the formation of the diagnosis, when it has taught us whether the pleural cavity be filled with fluid, solid, or semi-fluid matter; and in respect to this point, with proper care and attention, we shall always arrive at a satisfactory decision. According to Winterich,[1] the vocal fremitus in Cancer is oftener present than absent, whilst in effusion the reverse holds good.

But, if physical examination in some cases is at a loss to answer the questions proposed for diagnostic purposes, then the history of the case, the general appearance of the patient, the rapidity of development of the cancerous growth, the peculiar expression of the patient's

[1] Winterich's Krankheiten der Respirations-Organe, in Virchow's Pathologie und Therap Erlangen, 1854.

face, the peculiar tint of his skin, and perhaps the co-existence of Cancer in other organs, will sufficiently make up for the deficiencies of physical signs, and place us in a position which will enable us to make the diagnosis certain.

2. *Tubercular Infiltration.* — The physician will only be called upon to decide between phthisis and Cancer, when the affection has assumed great proportions. In this case it must be remembered that the latter disease never spreads so extensively as the former does, in which the total absence of rhonchi may also be an important sign. Hæmoptysis is a comparatively rare occurrence in Cancer, but not so in phthisis. The absence of the phthisical habit, the fact that patients suffering from Cancer are not unfrequently in a comparatively good condition, even in advanced stages of the disease, the co-existence of tumours, or the former removal of such, together with—sometimes lancinating—pain in the chest, and the microscopical examination of the sputa, whereby the product of Cancer sometimes may be found, will afford valuable diagnostical hints. Compression of the œsophagus, displacements of neighbouring organs in an extremely high degree, the rare occurrence of caverns, symptoms of compression of the aorta or vena cava, the not unfrequent limitation of the disease to one side only, are signs frequently met with in Cancer.

Diagnosis will become still more difficult, or entirely impossible, in cases of co-existence of tuberculosis and Cancer. It was due more particularly to Rokitansky that the opinion became general that tuberculosis and Cancer exclude each other, *i.e.* that they never do co-exist in the same person. Rokitansky, however, afterwards altered his opinion, saying that the co-existence of both diseases is merely a very rare occurrence. Other authorities hold the same opinion. But many cases have been published, showing that Cancer by no means excludes tuberculosis. I refer the reader to Dr. Pollock's case,[1] published in the "Transactions of the Pathological Society," and to a highly interesting one, recently published by Professor Friedreich,[2] concerning a woman forty-nine years of age, who suffered from primary Cancer of the left lung, with metastatic depositions in the heart, kidneys, suprarenal capsules, right lung, and pancreas, and from cancerous pleurisy of the left side. At the same time obsolete and recent tubercular enterophthisis and œdema of the brain were found at the post-mortem examination.

3. *Aortic Aneurism.*—In the course of development of cancerous affections, particularly at the root of the lung, great bulging in the clavicular region may take place, accompanied by pulsation and other symptoms resembling aneurism of the aorta. Here the remark of Stokes is of great value, concerning the contrast between the area of dulness on percussion and the pulsation. But the pulsation itself is of a different character in the two diseases, viz. circumscribed in aneurism, but diffused, not culminating in a particular spot, in

[1] Transactions of the Pathological Society, vol. iii. (1851-52), p. 254.
[2] Virchow's Archiv, xxxvi. 4, 1866.

Cancer, in which affection the ordinary signs of aneurism, as murmur or pulsation over the dull part, murmur above the clavicle, or propagated to the vessels of the neck, are also absent. I am furthermore inclined to believe that, in some cases, the sphygmograph will render great service in arriving at a decision, whether a disease be intrathoracic Cancer or aneurism. Gordon,[1] Martin Solon,[2] and others, have published very instructive cases, in which Cancer was mistaken for aneurism, and the treatment of Valsalva adopted. But, notwithstanding these authorities, I maintain that a careful examination and consideration of all symptoms, together with the history of the case, will seldom fail to result in a strict diagnosis, and to screen us from erroneous conclusions.

PROGNOSIS AND TREATMENT.—Cancer of the Lungs is a deadly disease, and, in spite of all medical efforts, leads finally to a fatal end.

The first symptoms, as a moderate pain in the chest, difficulty of breathing, a dry cough, &c., sometimes last for years without alarming the patient, till more severe and dangerous phenomena make their appearance, and with tremendous speed hurry the patient into the grave.

In the present state of our science we have neither means for extinguishing an existent cancerous cachexia, nor for causing deposits to be absorbed, which, once produced, seldom remain stationary for any long period, but go on increasing, destroying the affected tissues, and interfering with neighbouring organs.

In the good olden times, when physicians fancied that even a disease like Cancer would fly before a long prescription, many formulas were in vogue in which arsenic was the principal drug. This remedy was considered a specific, and eminent practitioners speak of it in terms of high commendation.

Others again advocated the use of conium, bichloride of mercury, the preparations of iron, and a number of other medicaments. But it appears that the efficacy of all these "specifics" became weaker and weaker in the same proportion as diagnostic science became strict and exact, and that arsenic and the other drugs effected a cure in those cases only in which a closer examination demonstrated that the case for which it had been applied was not Cancer at all.

But, though medical science has not yet arrived at a point to furnish us with means of *curing* Cancer of the Lungs, we must not rest quiet and leave such patients to their fate. Our profession has other tasks to fulfil where cure is impossible, namely, to relieve pain and alleviate other bad or dangerous symptoms, and thus to prolong life. In this respect we can act sometimes with very great benefit towards the sufferer.

I had a patient under treatment who dreaded the approach of night, this being for him the signal of excruciating pain, restlessness, and

[1] Med.-Chirurg. Transact. vol. xiii.
[2] Archiv. Gén. de Méd. tome xxiv. p. 142.

torture, during which he incessantly offered prayers to Heaven for his death. Besides deposits on his lungs, there were likewise some in his liver, and the stomach was also affected, and rejected food and medicines as soon as they were taken. When he came under my care, I injected, every night, half a grain of morphia hypodermically, and from that time he enjoyed at least good rest at night.

Our attention will therefore entirely be directed towards troublesome symptoms, improvement of the patient's nutrition, and keeping up his strength. Hence it becomes evident that bleeding in any shape and to any extent should only be resorted to in cases of pressing emergency. Dry-cupping, however, will prove beneficial when dyspnœa becomes troublesome, in which cases other counter-irritants may also be applied to the skin with success. In one case, under my care, a hot bath of a minute's duration gave rest to the much-exhausted patient, while other remedies failed to diminish the dyspnœa.

For the relief of pain, connected with Cancer of the Lungs, I can strongly recommend the hypodermic injection of morphia, beginning with a quarter of a grain, and increasing the dose according to the requirements of the case. In respect to the method of injection and mixing the solution, I refer the reader to my paper " On Hypodermic Injections," which has been published in the " Medical Mirror" of 1866.

Cough is another symptom which often resists all therapeutic endeavours. Where medicines can be taken, we should apply narcotics, opium, hyoscyamus, Indian hemp, and similar drugs. But, unfortunately, in many cases the stomach, either by reflex action or by being also affected by the disease, rejects the drugs, and renders our efforts useless. In these cases I propose the application of atomized fluids, which, indeed, would be the only means by which to introduce medicaments into the system. I refer the readers who are not acquainted with this mode of treatment to my work " On Inhalation." [1]

The patient's strength will appropriately be kept up by nutrient, easily digestible food, and avoiding everything which could possibly produce a conflux of blood towards the internal organs. In those unfortunate cases in which pressure on the œsophagus prevents the patient from taking solid food, it must, of course, be given as a fluid, and, if necessary, by the aid of the stomach-pump.

In the patient's room, a moderate but equal temperature ought to be kept by day as well as by night ; all the natural functions must be regulated as far as possible, and moderate exercise in the open air should be encouraged on fine days, and avoided only when it causes difficulty of breathing.

Some physicians advocate cod-liver oil. It may be tried in cases in which it does not at all interfere with the function of the stomach, but it ought to be given up at once if it causes loss of appetite or sickness.

[1] On Inhalation as a means of Local Treatment of the Organs of Respiration by means of Atomized Fluids and Gases, by H. Beigel, M.D. London : Hardwicke, 1866.

Fœtid breath, sometimes of unbearable intensity, disgusts not only everybody in the patient's room, but even the patient himself. This disagreeable quality of the breath can be destroyed in a short time, by inhalation of liquor. chlori, perchloride of iron, or creosote.

Should one be called upon to give some prophylactic hints to persons descended from parents who died of Cancer, the first care to be taken will be strictly to regulate the diet of such persons. Let them take regular exercise and live in mild climates, in places situated as high as possible; advise them to undertake voyages, or to undergo a course of the so-called "grape-cure," of which many physicians speak in commending terms, and which produced very good effects in a case under my own care.

The coast of England is a very healthy abode during the summer months for delicate individuals. But for such persons as wish to go abroad, Marseilles, Spezzia, Nice, Livorno, Venice, Heligoland, Kiel, Swinemünde, and the very pleasant isle of Rugia, could be recommended.

Places where grapes are methodically used for medical purposes are Meran in Tyrol, Dürkheim and Bingen in Germany, Krems in Austria, and Presburg in Hungary. The best time at which to send patients there is during the vintage, which is generally in the months of September and October.

PNEUMONIA.

By Wilson Fox, M.D., F.R.C.P.

SYNONYMS.—Peripneumonia,[1] Peripneumonia Vera (as opposed to Peripneumonia Notha, or Capillary Bronchitis); Febris Pneumonica, Hoffmann; Fièvre Pneumonique, Fluxion du Poitrine (French authors); Pneumonites, auct. var.

VARIETIES AND OTHER SYNONYMS. — Croupous and Catarrhal Pneumonia (*Rokitansky and modern German authors*). Acute Sthenic Pneumonia—Broncho-pneumonia (*English and foreign authors, signifying a similar distinction of origin and course*). Lobar Pneumonia— Lobular or Disseminated Pneumonia (*signifying anatomical differences in the extent and characters of the pulmonary affection*). Acute Pneumonia—Chronic or Interstitial Pneumonia (*signifying differences in course and duration, and also in anatomical characters*). Interlobular Pneumonia (*an affection of the interlobular tissue*). Primary Pneumonia—Secondary Pneumonia (*signifying differences in origin*). Other varieties have been termed, according to the origin or characters of the disease—Bilious, Gastric, Typhoid, Latent, Intermittent, Hypostatic, Tubercular, Scrofulous, Rheumatic, Gouty, Puerperal, Metastatic, and Pneumonia Potatorum (Huss).

ACUTE PNEUMONIA.

DEFINITION.—A disease whose essential anatomical feature consists in the inflammation of the vesicular structure of the lungs, which is thereby rendered impervious to air through the accumulation in the interior of the alveoli of the products of such inflammation. Clinically it is characterised by pyrexia, which, in the majority of cases, when the disease is primary, commences with rigors; it is also commonly attended by pain in the side, by dyspnœa, cough, sanguinolent sputa, great physical prostration, and by the physical signs of pulmonary consolidation. Its course, when primary, is usually acute, and tends to terminate favourably by a crisis occurring from the third to the tenth day, but it may prove fatal from the first to the fourteenth day, or at later periods. When secondary to other diseases,

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[1] Grisolle considers that the prefix περι is merely expletive.

the termination by crisis is uncommon, and its duration is also more protracted; and under all circumstances of its origin it may, in some instances, lapse into the chronic state. Its immediate cause is uncertain, and it appears in the majority of instances to depend either on an unknown but suddenly produced dyscrasia, or on an alteration in the composition of the blood induced by various diseases. In other cases it is produced through the extension to the pulmonary tissue of bronchial inflammation, or it may originate through local disturbances of the pulmonary circulation occasioned by congestion or collapse, or by obstruction though emboli of the pulmonary artery, or it may be caused by mechanical injury to the tissue of the lung.

Although the anatomical characteristics of Pneumonia can be defined with a certain approach to accuracy, the clinical features of the disease may nevertheless present a considerable diversity of aspect under the varied circumstances of its origin.

In some cases variations in the anatomical process may be observed corresponding with these different features of the disorder, but distinct lines of demarcation are in this respect very frequently wanting, and the author believes that the anatomical distinction between the " croupous "[1] and the " catarrhal " forms, on which especial stress has of late been laid, is by no means so sharply defined as some recent writers have maintained.

From a clinical point of view, however, the separation of the main types of these two forms of the disease into distinct species has a practical value, and it may therefore be stated that the principal classes to be distinguished are (1), Primary or Acute Sthenic Pneumonia; (2) Secondary Pneumonia, including most of the catarrhal forms; (3) Interlobular Pneumonia; (4) Chronic Pneumonia. Under the head of Etiology, the relations of the different forms of the acute disease will be treated collectively.

HISTORY.—In the earlier days of medicine, since the times of Hippocrates and Galen (by whom, however, both diseases were recognised), Pneumonia was confounded with Pleurisy to such an extent that the rusty sputa characterising the former disease were described as an attribute of the latter; and pleurisy was said to be capable of producing cavities in the lung. Valsalva, Morgagni, Huxham, and Boerhaave gave accurate descriptions of Pneumonia, but still the

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[1] The term " Croupous," introduced by Rokitansky, and largely used in Germany, appears to the author to be in some respects best avoided. It was originally employed by Rokitansky to define a particular form of exudation, and in its application to Pneumonia he drew a parallel between this disease and croup of the larynx, attended by false membrane. The analogy appears to be an erroneous one in two aspects, for in the first place the Pneumonia attending laryngeal diseases when false membranes are present is seldom seen in the form recognised as characterising acute sthenic Pneumonia, but is most commonly of the type termed Broncho-pneumonia; and, secondly, there is no boundary line of distinction between the forms of the disease characterised by a coagulable exudation in the vesicles, and those where cell-products are mingled with some fluid exudation. The extreme types are, it is true, distinct, but every shade of gradation may be observed between them.

distinction between it and pleurisy was not completely recognised until the writings of Bichat and Pinel, and the collapse of the lung attending pleuritic effusion was by most other writers mistaken for inflammation of its substance.[1] The accurate clinical separation of the two diseases was finally fully evolved by Laennec.   Since his time, the most important advance in the definition of the disease has been that made by Jörg, Bailly, and Legendre in the separation and distinction of the various forms of collapse, or defective expansion from true inflammatory action.   The other features of interest in recent researches will be alluded to in their appropriate places.

ETIOLOGY.—On many points in the etiology of Pneumonia the only data at our disposal refer to the disease as a whole, irrespective of any of the special varieties before alluded to.   The circumstances predisposing to particular forms will be, as far as these are known, described separately.

A. *Race and Climate.*[2]—Inflammation of the lungs appears, with but few exceptions, to be more commonly associated with climates presenting marked and rapid variations of temperature than with extreme degrees of either cold or heat.   Thus in tropical climates it is uncommon during the continued hot seasons, and, on the other hand, in some of the expeditions to the North Pole the disease has been almost unknown.   It is said also to be very rare in Iceland.   Throughout the European continent, below 60° north latitude, it is a very prevalent disease, and the southern portions, including the shores of the Mediterranean,[3] are nearly as liable as the more northern countries. Thus in Copenhagen the mortality from Pneumonia is 6·3 per 100 of all deaths; and in Gibraltar 41 per 1,000 soldiers suffer from the disease.   In the more tropical climates, elevation above the sea-level increases the frequency of the disease, and it is very common in the high table-lands of Mexico.[4]   The disease appears to be rare in Egypt,

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[1] According to Pinel, " Nos. Philos." ii. 145—191 et seq., the question of the distinction between these two diseases appears to have given rise to the most animated discussion among the writers of the 17th century.   The history of the earlier views on Pneumonia will be found at length in Grisolle's work on Pneumonia; also in Wunderlich's " Path. Therap." art. Pneumonie, and in Neumann, "Krankheiten des Menschen," 2e Ed. i. 151 (quoted by Wunderlich).   The confusion between Pneumonia and pleurisy was aided by the fact, that before the writings of Bichat the term pleura was limited to the parietal membrane, the visceral portion being confounded with the tissue of the lung.

[2] For a large number of the data under this head, the author is indebted to the writings of Grisolle, "Traité de la Pneumonie," and Hirsch, " Handb. der Hist. Geograph. Pathol." 1864 ; and also to an elaborate statistical work on the Geographical Distribution of Pneumonia, by Ziemssen, "Monatsblatt für med. Statistik und offene Gesundheits-pflege," 1857, analysed at considerable length in Canstatt's " Jahresb." 1857, ii. 119.   Many of the data on this subject refer, however, to pleurisy and Pneumonia collectively, and this is especially the case with those given in Hirsch's work.

[3] Clark on Climate, p. 121.

[4] Elevation in cold climates, in some situations, also appears remarkably to predispose to the disease.   Thus of the French troops quartered on Mont Cénis from December to May, one-fourth of the whole number were attacked by Pneumonia. (Chomel, Leç. Clin. Méd., Ed. Sestier, p. 451.)

though bronchitis is common in the valley of the Nile; in India it is more common in Bengal than in Bombay. Though equability of temperature appears to confer a certain degree of immunity from the disease, yet there are some remarkable exceptions; for in Senegal, which possesses a variable climate, Pneumonia is rare, while in the Bermudas, where the temperature is remarkably uniform, it is by no means uncommon; and it is stated, on the authority of Dr. Farry,[1] that Pneumonia and affections of the lungs in general are less common, both in the Northern and Southern States of the Union, than in the central portions where the temperature is more uniform. Oregon and California appear to enjoy a singular immunity from the disease. In certain countries, as in Sierra Leone, the Cape, and the Mauritius, the negro races, at least when employed in military service, appear to suffer more than the whites; but it is considered possible that the preponderance of the affection among them is due to their being more exposed to vicissitudes of temperature than the European soldiers, with whom greater precautions are taken.

The disease is said to be more common among sailors on land than when at sea;[2] but it may be questioned whether this difference is not in part due to other influences, causing an increased relative frequency on land, such as greater irregularity of life and severer exertion.

In England, Pneumonia appears, from the returns of the Registrar-General for 1863-4, to rank next after the following main causes of mortality :—Phthisis, bronchitis, scarlatina, old age, and convulsions. The frequency, and also the mortality of the disease, however, vary considerably in different years, as is shown by the contrast of 26,052 deaths registered under this head in 1855 when compared with 21,118 occurring in 1867;[3] and the data of nearly all the large hospitals of the Continent furnish confirmatory evidence of the same kind.[4]

It would appear from Ziemssen's analysis that the mortality from Pneumonia is greater in large towns than in country districts; but in this respect there are considerable differences in degree between different cities, that of Cork being 0·5; London, 1·7; Paris, 2·3; Turin, 3·8; and Algiers, 4·3 per 1,000. Ireland seems to suffer to a less degree than most of the European countries.

B. *Classes and Professions.*—There appears to be a general con-

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[1] American Journ. Med. Science, 1841. (Grisolle.)

[2] Dr. Wilson's report to the Admiralty gives for 1,000 sailors : Short voyages, 29 per 1,000 ; home service, 35·1 per 1,000 ; Mediterranean, 31·8. Sailors as a class suffer but little—175 per 24,000. (Le Roy de Méricourt.) These data are quoted from Grisolle.

[3] In the last-named year these proportional numbers are 995 deaths from Pneumonia, to 1,000,000 living ; and 45,275 to 1,000,000 of deaths.

[4] This is especially evident from the statistics of Huss, "Behandlung der Lungen Entzündung :" for while the average number of cases during 16 years was 163·5, these in 1849, 1851, and 1853 amounted respectively to 243, 242, and 203 admitted to hospital ; while in 1840, 1841, and 1844, the numbers were only 107, 102, and 97. It will be seen in the section devoted to the prognosis that the mortality of the disease in different years also presents considerable variations ; and also that the relative mortality at different seasons by no means corresponds to the frequency of the disease at these periods.

sent that Pneumonia is more common among the labouring than in the wealthier classes of society, and that, among the former, those whose occupation involves the severest exertion and the greatest amount of exposure are the most liable to suffer. In the English army the soldiers suffer more than the officers.[1] The disease is more common in the French army than among the civil population.[2]

C. *Seasons.*—It may be stated as a general truth, that in European countries Pneumonia is most common during periods of the year in which there are the greatest vicissitudes of temperature, while either a continuously low or high temperature has much less influence in its production. Thus, of 2,616 cases collected by Huss[3] during a period of sixteen years in Stockholm, the spring months, March, April, May, and June, gave 49 per cent.; the winter months, November, December, January, and February, yielded 30 per cent.; and the summer months, July, August, September, and October, 21 per cent. Of the individual months, August and September are those in which the greatest immunity is observed, but this is nearly equalled by June and July, while April and May show the greatest frequency. Huss states that the relative frequency in individual months in different years corresponds closely to rapid changes of temperature observed in them. Barometric variations, independently of the influence of wind, appear to have little or no effect in the production of the disease. The converse, however, appears to hold true of cold winds, and particularly of those from the north and east; and though the effects of these in the production of Pneumonia have been more observed in the aged, and also, though to a less degree, in the young, than in persons of middle life, yet there is a strong probability that their agency is similarly exerted at all ages.[4] It was stated by Huxham[5] that dry cold air was most frequently associated with Pneumonia of an inflammatory type, and that "bastard peripneumonies" were most common in damp seasons. Dr. Jackson[6] has also shown that in

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[1] On the Mediterranean stations the soldiers suffer from Pneumonia in the proportion of 32 to 42 per 1,000; the officers in the proportion of 14·1 per 1,000. On the Canadian stations the proportion of soldiers affected is 43 per 1,000, and that of the officers is 10·6 per 1,000. (Quoted from Grisolle.)

[2] Deaths from Pneumonia in the civil population of France, 30 per 1,000; in the army, 39 per 1,000. (Lancereau, Ann. d'Hygiène, 1860, xiii. 269. Valleix.)

[3] The amount of statistical evidence on this head is large and conclusive, and the results obtained by all observers agree very closely with those of Huss. For other references see Chomel, "Lec. Clin. Méd. 'Pneumonic,'" p. 444; Grisolle, loc. cit., 139; Wunderlich, "Allg. Path. Therap." Bd. iii., Abth. ii. B., p. 304; Bamberger, "Wien. Med. Woch." 1857; Roth, "Würzb. Med. Zeitsch." 1860; Hamernigk, "Die Cholera Epidem.," Prag. 1850. Ziemssen, "Die Pleuritis und Pneumonie im Kinderalter," p. 187, found in Grieswald a rather larger proportion during the summer months than has been noticed by other observers. He attributes this to the cold winds and rapid variations of temperature observed there during this season. Morehead, "Dis. of India," pp. 300—303, found Pneumonia in India to be most common in the cold season, and next in frequency in the wet season. During the latter period it is very liable to be complicated by intermittents.

[4] See for evidence on this subject Grisolle, p. 142.

[5] Essay on Fevers, 1757, p. 222.

[6] Dr. Sibson, Brit. and For. Rev. 1858, xxii. p. 23.

Massachusetts, a damp climate, complications are more common than in drier atmospheres.

D. *Age* must be regarded as an important etiological element in the predisposition to Pneumonia, and it is also one of the conditions most materially influencing its mortality.

Some of the details given by writers antecedent to the researches of Legendre and Bailly are, however, unreliable, owing to the confusion then existing between Pneumonia and collapse of the lungs occurring in infancy. Thus Valleix and Vernois[1] stated that of 114 newly-born children 113 had hepatization of the lungs. In spite of these doubts, however, there is very little question that Pneumonia is a very frequent disease of early life. Of 186 cases of primary acute (croupous) Pneumonia in children, recorded by Ziemssen,[2] 117 occurred in the first six years of life, and only 69 in the succeeding ten years. Gunsburg,[3] for 5,000 cases of Pneumonia, gives the following relative table of frequency at different ages :—

	Years.		Years.			
Under	$1\frac{1}{2}$			. .	11	per cent.
From	$1\frac{1}{2}$	to	14	. .	13	,,
,,	14	,,	20	. .	6	,,
,,	20	,,	30	. .	17	,,
,,	30	,,	40	. .	16	,,
,,	40	,,	50	. .	10	,,
,,	50	,,	60	. .	9	,,
,,	60	,,	70	. .	7	,,
,,	70	,,	80	. .	11	,,

Lombard has given, further, the following proportion of deaths from Pneumonia and deaths from other diseases at different ages :—

Deaths from all causes.			Age. Years.		Years.		Pneumonia.	
274	. .	under	$1\frac{1}{2}$			. .	56	$= \frac{1}{5}$.
310	. .	from	$1\frac{1}{2}$	to	14	. .	70	$= \frac{1}{4}$.
112	. .	,,	15	,,	19	. .	3	$= \frac{1}{37}$.
387	. .	,,	19	,,	27	. .	39	$= \frac{1}{10}$.
766	. .	,,	27	,,	75	. .	46	$= \frac{1}{16}$.

Grisolle's statement may therefore be regarded as embodying the truth on this question, viz. that Pneumonia (both primary and secondary, lobar and lobular collectively) is a disease very frequent in infancy, that it is less common from infancy to twenty years of age, that it is comparatively frequent from twenty to forty, less so from forty to sixty, and very frequent, and also very fatal, after sixty years of age. To this it may further be added, that the Pneumonia of old people and of children approximates more, but by no means exclusively, to the type of catarrhal, or broncho-pneumonia.

E. *Sex.*—In the Pneumonia of adult life, males are more commonly affected than females in proportions varying from two or three to one.[4]

[1] Valleix, Clin. des Malad. des Enfans nouveaux-nés, 1838, p. 114.
[2] Loc. cit. p. 155.
[3] Klinik der Kreislaufs und Athmungs-Organe (Breslau, 1856), quoted from Huss, loc. cit.
[4] The proportion of 2 males to 1 female is that given by Grisolle and generally accepted. Of the actual numbers treated by Huss, the proportion was 5 to 1, but it

This difference between the sexes is not observable in the earlier periods of life;[1] but it becomes apparent first at ages when the occupations of the sexes differ, and when males are more exposed to climatic influences than females. When, however, the conditions of life for both sexes are identical, this relative disproportion in great measure disappears.[2] Huss has adduced the fact that it is also much less marked in advanced age.[3]

Females, as it would appear from Grisolle's data, are somewhat more predisposed to the occurrence of the disease at the menstrual period. Neither pregnancy nor the puerperal condition seem, however, to create any special proclivity, except when the latter is complicated by septicæmia.

F. *Constitution.*—Opinions differ whether primary Pneumonia most commonly attacks the vigorous or those in previously bad health. The Hippocratic doctrine was in favour of the former view, which is also supported by Grisolle. Huss, on the contrary, thinks that it is more common in weakly subjects. Dr. Hughes Bennett,[4] in 118 cases—84 males and 34 females—found that of the males 27, and of the females 22, were in bad health at the time of the seizure. Huss considers that the fact that robust males are frequently attacked depends in great measure on the greater degree of exposure to external influences to which they are subjected. Chlorotic females seldom suffer. Rickets, on the other hand, appears to produce a predisposition to the disease, for of twenty-four patients dying rickety, Grisolle found Pneumonia in one-half. It is possible that this may be caused by the greater severity of bronchitis and the increased tendency to collapse in these subjects, and also to the fact that collapse of the lung when complicating bronchitis induces a liability to further inflammatory changes.

It has been observed that some persons are liable to repeated attacks of the disease—a peculiarity which may either be due to some special but unknown constitutional predisposition, or to the fact that previous attacks induce a proclivity to its return. The latter hypothesis is to some degree favoured by the fact that the lung first

amounted to 3 to 1 when calculated on the total numbers of all cases of males and females admitted to hospital. The proportion in the general hospital at Vienna (quoted by Huss) is 1·98 males to 1 female. Huss thinks that the greater disproportion observed in the more northern climate between males and females may be due in part to the greater intensity of climatic conditions to which the former are there exposed.

[1] Ziemssen, in 91 cases of children under four years of age, found that the boys affected numbered 41, and the girls 35.

[2] Thus Tolmouche has observed that in prisons the number of individuals of the two sexes suffering from Pneumonia are, comparatively speaking, equal (Ann. d'Hygiène, xiv. pp. 25—27. Ruef also (Heidelb. Med. Annalen, ii. 1836) has noticed a similar equality in the liability of the sexes to the disease when women are employed in outdoor labour.

[3] Of the cases between the ages of 16 and 50, the males formed 85·5 per cent., and the females 14·5 per cent.; but of the cases between 50 and 70, the males constituted only 55·19, and the females 44·81 per cent. Dinstl also (Oest. Zeitsch. für prakt. Heilkunde, viii. 1862) found in 1,212 cases of Pneumonia, that after ætat. 50 the number of females affected was greater than that of the males.

[4] The Restorative Treatment of Pneumonia, 1866, p. 24.

affected is the most liable to suffer in a subsequent attack.[1] In 175 cases analysed by Grisolle, 54 had suffered from previous attacks, but of these only two were in females. The period between the attacks varied from one month to twenty-five years. Most usually the intervals varied from three to five years; but these tend to become shorter in proportion as the attacks become more frequent.[2]

The number of attacks from which individuals have suffered is also very remarkable. Thus Andral[3] records a case of a patient who had had fifteen attacks in eleven years, Chomel[4] has seen ten recurrences, J. P. Frank[5] eleven, and Rust has even recorded twenty-eight attacks in the same individual.[6] Intermittent fever also predisposes to recurrence. A patient of Ziemssen's thus affected had four attacks in five years, three of which were in the left lower lobe and one in the right upper lobe.[7]

Difficult dentition predisposes to Pneumonia in children,[8] and also makes the prognosis more unfavourable. Favourable hygienic influences confer a certain degree of comparative immunity from the disease. Drunkenness appears to act powerfully as a predisposing cause of Pneumonia, though its effect in immediately producing the disease may be regarded as somewhat doubtful.

G. *Direct Exciting Causes.*—The influence of these in the production of the acute primary disease has been very variously estimated by different observers. Some authorities, and particularly writers of the last century,[9] attribute its origin mainly to the influence of a chill — an antecedent which others have denied from statistical data. Grisolle asserts that a discoverable cause of this nature could only be affirmed in one-fourth of his cases. Chomel[10] and Andral[11] express very similar opinions. Ziemssen says that among children a discoverable cause only existed in one-tenth of his cases. In fifty-three

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[1] In 35 cases of recurrence collected by Grisolle, the return of the disease was noted 25 times in the lung first affected. In the remaining 10 the disease changed sides : Pneumonia of the left lung recurred more frequently than that of the right, in the proportion of 16 to 9. This is the more remarkable when it is remembered how much more frequently the right lung suffers from the primary disease.

[2] Dr. West, of 78 cases in children, found that 31 had suffered from previous attacks. Of these, 21 had been affected once, 4 twice, and 2 four times, and 4 others were said to have had several attacks. Ten of these patients were under 2 years of age ; ten more between 2 and 3, and the remaining 11 were between 3 and 6. Ziemssen, in 201 cases of children, found 19 cases in which the attacks were repeated. Of these, 14 had Pneumonia twice, 3 three times, and 2 four times. In some instances the disease recurred at corresponding periods of consecutive years.

[3] Clin. Méd. iii. 371.   [4] Dict. de Méd. xviii. art. "Pneumonie."

[5] Interpretationes Clinicæ, Tubingæ, 1812, p. 96. (Grisolle.)

[6] Quoted by Dr. Williams, art. "Pneumonia," Cyc. Pract. Med. iii. 406.

[7] Ziemssen, loc. cit. 154.

[8] Of 201 cases of Pneumonia observed by Ziemssen, this condition was present in 37. Of these, 16 had Broncho-pneumonia after long-continued bronchitis, and 21 suffered from primary or "croupous" Pneumonia.

[9] Pinel (Nos. Phil. ii. 163) defines as the causes of primary Pneumonia : "Impression brusque d'un air froid après un violent exercice, comme la course, la lutte, le chant, les cris, une équitation rapide contre la direction du vent, une boisson froide lorsqu'on est échauffé."

[10] Leçons, p. 464.   [11] Clin. Méd. vol. iii.

cases analysed by myself, a distinct cause, which when present was always of the nature of a chill, could only be affirmed in sixteen. It must, however, be admitted that this is the most common of the discoverable causes, and that the frequent absence of evidence of such an origin is common not only to Pneumonia, but also to many catarrhal affections, and further to acute rheumatism, diseases which, to say the least, are very frequently due to this immediate agency. The most probable explanation of such cases would appear to lie in the existence of a more extreme constitutional susceptibility, in consequence of which causes so slight as to pass unnoticed at the time of exposure may produce effects which persons less predisposed to suffer from their influence would have escaped. I do not think, as far as my own observation has gone, that the cases excited by a chill can be separated from the rest and placed in the category of Bronchopneumonia, for in most of the instances coming under my own cognizance these cases have run as typical a course of acute primary Pneumonia as those in which no such cause has been discoverable. The indirect evidence afforded by the seasons of the year at which Pneumonia is most prevalent, strongly bears out the opinion that vicissitudes of temperature are among the most important agencies in its production. They appear to act most strongly at the two extremes of life. Cruveilhier[1] particularly noticed the injurious effects of cold on the aged in the Salpêtrière; and Hourmann and Dechambre,[2] out of 156 cases of Pneumonia in old people, observed 140 in the winter and early spring months, from November to May. Both these writers, and also Cruveilhier, remark upon the injurious effects of north and north-east winds in producing inflammation of the lungs in the aged.[3]

Laennec thought that prolonged exposure to cold had more effect than a sudden chill, but I cannot say that my own experience has led me to adopt this view. Nearly all the cases of Pneumonia which I have observed from traceable causes were owing to a temporary chill, such as a wetting, exposure to draughts of cold air when heated, and similar influences. There can be very little doubt but that Pneumonia, in many instances at least, must depend in great measure on predisposing constitutional or local conditions, whose nature is unknown, but whose influence is distinct. It is to their influence that the special localization of acute diseases arising from general in contradistinction to specific causes, is due; and it is also to the greater or less degree in which they predominate, that the relative facility of the production of such diseases may in great measure be attributed.

Excessive exertion appears to act as an occasional cause. Wunderlich quotes a statement of Barth's to the effect that he had traced

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[1] Anat. Path., liv. xxix.
[2] Pneumonie des Vieillards, Arch. Gén. 2ᵉ Sér., xii. p. 29.
[3] The mode of action of these causes will be further considered under the head of Pathology.

this cause in 12 out of 125 cases, and Wunderlich says that he can confirm Barth's experience.

Traumatic causes do not easily produce a pneumonia of any extent or severity: the lung appears to have remarkable powers of recovery from direct injury.[1] Injuries and blows to the chest are however occasionally followed by Pneumonia without distinct evidence of direct laceration of the lung.[2] The mechanism of such influences appears in some cases very obscure. Thus in a case admitted into University College Hospital, under Sir W. Jenner, a patient struck his shoulder-blade on rising from a stooping position. He had previously been in apparently good health, though on admission he was found to be suffering from albuminuria, in addition to signs of pleuro-pneumonia on the side struck. Pericarditis also supervened, and the case proved fatal. The pneumonia was in the lower portion of the upper lobe, and there was also extensive pleuritic effusion on the same side, but there was no evidence of mechanical injury to the chest-wall or to the lung. The kidneys were fatty. It is probable that in this case the pre-existing kidney disease acted as a powerful predisposing cause to the pathological conditions found.

Pneumonia may, on the other hand, be easily excited by foreign bodies entering the lung from the bronchi. This condition is said to be not uncommon in those cases of dementia when food finds its way into the bronchi, and where gangrene of the lung is very liable to supervene. Grains of wheat or beards of barley entering the bronchi are also occasional causes of Pneumonia.[3] Blood gravitating into the vesicular structure of the lungs in cases of pulmonary hæmorrhage may occasionally act as an exciting cause,[4] and it is thought probable that the disseminated Pneumonia observed in diphtheria and capillary bronchitis may be, in part at least, occasioned by the gravitation or insufflation into the air-vesicles of the fluid secretions of the bronchial tubes.

It is very doubtful whether irritating vapours can produce true lobar Pneumonia. They may, however, produce a disseminated form

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[1] Grisolle, pp. 43-4.

[2] See a case quoted by Grisolle, loc. cit. 316, from J. P. Frank, of a porter who had overstrained himself; also Duchek, "Abtheilungs-bericht Allgem. Krankenhaus zu Prag.;" Prager Vierteljahresch. 1853, xxvii. p. 37 — two cases where Pneumonia followed a blow on the chest; also Wunderlich, loc. cit. Bd. iii. Abth. ii. 13; also noted by Morgagni, "Epist." ii.; also a case by Andral, "Clin. Méd." iii. obs. vii. p. 293.

[3] Grisolle, p. 146.

[4] See Dr. Hermann Weber's, Dr. C. Bäumler's, and Dr. Sanderson's papers in the Trans. Clin. Soc. iii. This subject is a very wide one, and involves the disputed question whether hæmoptysis, unassociated in the first instance with tubercles, can originate a disease running the course of phthisis. I have more than once seen Pneumonia follow hæmoptysis in the course of early phthisis, but I have hitherto regarded it as probable that the hæmoptysis may be the result of the congestion which precedes Pneumonia acting on the weakened pulmonary vessels. In some cases of phthisical subjects, this appears to be the undoubted mechanism of the hæmoptysis observed; but in other cases there is a strong probability that the Pneumonia results from the hæmoptysis in the manner described.

of the disease, resembling closely the "lobular pneumonia" occasionally occurring in bronchitis.[1]

H. *Epidemic Causes.*—The only positive data on this subject are those afforded during the prevalence of epidemics of influenza. This disease has certainly a considerable tendency to give rise to Pneumonia, which is for the most part of a catarrhal type. Thus Nonat[2] observed, during the epidemic of influenza in 1837, that of 300 deaths in the hospital Hôtel-Dieu, in Paris, in the month of February of that year, 80 were due to Pneumonia; and Laserre,[3] in La Pitié, observed in three months in 1842, during a similar epidemic, 31 cases of Pneumonia.

It would appear, however, that unhealthy conditions of crowding, with bad ventilation, strongly predispose to the disease when other causes, particularly measles, are present; and some evidence has lately been adduced to show that similar influences may operate independently of the presence of any immediate exciting cause.[4]

Griesinger[5] has stated that in malarial districts Pneumonia has at times a tendency to assume an epidemic character. It may be doubted whether, independently of such causes, Pneumonia can be considered as an epidemic due to a specific poison, or whether its greater prevalence at certain seasons, and in particular years, producing an apparent resemblance to a zymotic disorder, has not resulted from some of the atmospheric agencies before alluded to.[6]

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[1] See Bretonneau, Rech. Infl. Spec. Tiss. Muqueux, Paris, 1826, p. 100. Gendrin (Hist. Anat. des Inflam. ii. 302) says that if an animal be made to breathe chlorine, the lungs are found studded with little solid nodules arising from an exudation into the air-vesicles. Gendrin considered these to be identical with some forms of tubercle. It may also be recalled that Cruveilhier produced similar results (to which he attached the same interpretation) by injecting mercury into the trachea. Reitz (Sitzb. K. K. Akad. zu Wien, 1867; Math. Nat. Wissch. Cl. lv. 3) has varied these experiments by injecting caustic ammonia into the trachea. I have repeated this experiment in a dog. The result was an intensely developed membranous exudation, extending throughout the trachea and smaller bronchi, but becoming more fluid and puriform in the latter. There was no uniform lobar consolidation in the lungs, but these were studded throughout with small yellow spots, solid, not at all prominent, rarely exceeding the size of a pea, somewhat irregular in their outlines, finely granular on section, breaking down in various parts into cavities which in some places attained the size of a hazel-nut. These were filled with a diffluent puriform matter, and, when near the surface, they projected like blisters from under the pleura by which they were covered. Death had occurred on the third day after the experiment. Dinstl, however (Oest. Zeitsch. Prakt. Heilk. viii. 1862, and Schmidt's Jahrb. 1866), has occasionally seen Pneumonia arise from the inhalation of irritating vapours.

[2] Arch. Gén. de Méd. 3ᵉ Sér. tome ii. 1837, p. 16.   [3] Ibid. xv. 1842, p. 130.

[4] Thus Dahl, "Norsk Mag. für Laegevidenz," xxii. Hft. 6. Virchow's Jahresb. 1868, ii. 95, has twice observed an epidemic of Pneumonia in the prison of Christiania. The first of these was in 1847; the second in 1866-7, when of 366 prisoners, 62 had Pneumonia, or one-sixth of the whole number. The servants working outside were equally affected with the prisoners. In other years Pneumonia has been a rare event in the prison. Prof. Boeck, who was consulted by the Government, considered that overcrowding had a great influence in the production of the disease. In the "epidemic" of 1866-7, the weather was very cold during a great part of the prevalence of the disorder, and Pneumonia was common also in the surrounding district.

[5] Infections Krankheiten; Virchow's Handbuch, Sp. Path. Therap. ii. 43.

[6] The descriptions of epidemics of Pneumonia are only to be found in older writers, nd the nature of the disorder must in some of these cases be considered at least

It has been asserted that Pneumonia and "typhus" fever have a tendency to appear simultaneously, and it has hence been concluded that some connexion may therefore possibly exist between these diseases. This belief is disproved by the returns of the Vienna hospitals, and also by Huss's statistics; though Huss considered that during the prevalence of these disorders Pneumonia is liable to assume the typhoid form.

I. *Influence of other Diseases in the production of Pneumonia.*— There appear to be at least six categories under which Pneumonia occurring in the course of other diseases may be classified :—

1. It may be the immediate effect of the poison producing the primary disease, or of the altered composition of the blood thus induced,[1] and in this light it is probable that many of the pneumonias occurring in the course of the acute febrile diseases should be regarded.

2. It may be the result of accidental products accumulating in the blood, as is seen in albuminuria, and possibly in diabetes,—or it may arise from the mechanical or infecting influence of solid materials formed elsewhere, and conveyed by the blood current to the lungs, as in thrombosis and in some cases of pyæmia.

3. It may be the secondary result of other diseases affecting the lungs or air-passages, as tubercle or bronchitis.

4. It may be the effect of mere passive congestion, mainly of mechanical origin, arising either from valvular disease of the heart or from weakness of the circulation, aided by defective respiratory movement and dependent position, and in many cases by collapse of the lung in the course of some of the acute febrile and also in that of chronic exhausting diseases.

5. It may be the result of a direct extension of diseases affecting other organs, as when abscesses of the abdominal viscera communicate with the lungs. In some cases pneumonia, secondary to pericarditis, may have a similar origin.

6. It may be a purely accidental complication.

In many of the acute febrile diseases no other cause can be assigned for the occurrence of Pneumonia than the presence of a blood poison.[2] In others the mechanism is more complex, as in diphtheria

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doubtful. Lebert, "Path. Anat." i. 651, says, however, that he has convinced himself of the existence of epidemics of Pneumonia in certain parts of Switzerland. Further information on this subject may be obtained in the following works :—Hirsch, loc. cit. ; Ozanam, Hist. Méd. des Mal. Épidém., Paris, 1835 ; Lepecq de la Cloture, Obs. sur les Malad. et Consid. des Epidémiques, Paris, 1776-1788 ; Max Simon, Etude Pratique rétrospective, et comparée sur le Traitement des Epidémiques au 17e Siècle, Paris, 1859. (Quoted by Lebert, loc. cit.)

[1] O. Weber, in addition to other internal inflammations, has succeeded in producing diffuse Pneumonia by injecting the blood of a febrile dog into another healthy one. (Pitha and Billroth's Handbuch der Chirurgie, i. 610.) See also Virchow, Ges. Abhand. 660, et seq. Also Billroth, Archiv für Klin. Chirurg. vol. vi.

[2] It has been noticed by Andral that Pneumonia may appear with the first invasion of the exanthemata, and that its occurrence at these early periods sometimes coincides with an imperfect development of the eruption. In a case of variola he observed, during the invasion, crepitation in both lungs, with a viscous rusty ⸺ which

and measles, when the effect is probably in part due to the secondary
effects of bronchitis or collapse; and even in some cases of typhoid
fever Pneumonia may arise either from embolism,[1] or from secondary
blood-poisoning resulting from the ulceration of the intestines.

The characters also of the Pneumonia arising in the course of other
diseases vary considerably. In some, as in measles and hooping-
cough, it mainly presents the characters of lobular or broncho-pneu-
monia; in others, as in variola, the inflammatory changes may be
either lobar, or may be disseminated irregularly throughout the lungs.
The appearances in diabetes may be either those of the acute lobar form,
or the Pneumonia may occur in disseminated nodules, tending to
undergo a necrobrotic or cheesy change, and which appear to be closely
allied to, if not identical with, the tubercular process. In albuminuria
either the acute lobar form may predominate with firm exudation, or
the inflamed part may present a smoother section, together with softer
consistence and a more translucent appearance, arising from co-existing
pulmonary œdema.

Of the acute specific fevers, measles is that most commonly attended
by Pneumonia. The frequency of the latter disease varies, how-
ever, in different epidemics, and at different periods of the same
epidemic;[2] and it is a more common complication of the disease
during childhood than in adult life. Typhoid fever stands next
in order of frequency—typhus fever, according to the statement of
Dr. Murchison,[3] involving a minor degree of liability to the disease.
In both these diseases, however, the data are somewhat uncertain,
owing to the liability to hypostatic congestion of the lungs, which is
commonly found when they prove fatal. In typhoid fever especially,
the Pneumonia tends occasionally to assume the lobular and vesicular
forms of the disease.

In scarlatina, Pneumonia is less common during the earlier stages,
but is by no means rare when in its later periods it is complicated
by albuminuria.

In glanders and farcy, secondary Pneumonia is extremely common.
It usually assumes a disseminated form and tends to pass into suppu-
ration, presenting in this respect many features common to pyæmia.[4]

Pneumonia is occasionally observed in cases of erysipelas.[5] In

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vanished on the appearance of the eruption. I have recently seen a case where crepita-
tion and, dulness at the base of the lung disappeared within twenty-four hours after the
eruption of variola had taken place. (Cf. Clin. Méd. iii. pp. 409—460.)

[1] I have seen a well-marked instance of this, where both lungs contained infarcta
surrounded by secondary Pneumonia.
[2] Barthez et Rilliet, Mal des Enfants, iii. 264. These authors observed 65 cases of
Pneumonia and lobular Broncho-pneumonia in 167 cases of measles. Bartels (Virch. Arch.
xxi. pp. 75-6), in an epidemic in 1860, found Pneumonia or Broncho-pneumonia in 12 per
cent. of his cases, but these complications contributed 80 per cent. of the deaths which
occurred.
[3] Continued Fevers, p. 184.
[4] It is remarkable that, although this is the condition most ordinarily found in the
human subject, yet that in the horse these diseases are associated with peri-bronchitis,
and in this respect closely resemble tubercle. (Cornil and Ranvier, Manuel Histol. Path.)
[5] Stokes, Dis. of Chest, p. 339.

some cases it appears to be due to secondary blood-poisoning, and to assume the disseminated form of pyæmic Pneumonia.[1] In other instances, however, it appears to be rather of the nature of an intercurrent phenomenon, and approximates more or less closely in its characters to those of the acute primary disease; and it appears not improbable that the Pneumonia may, under these circumstances, originate from the same blood-poison as that which gives rise to the erysipelas. It has also been observed to arise by a propagation of the inflammatory action from the skin, extending through the mouth, fauces, and air-passages to the lung-tissue.[2]

In the course of acute rheumatism, Pneumonia is a not very uncommon complication.[3] In some cases it appears in a form truly metastatic with the rheumatic affection of the joints;[4] but most commonly the joint affection persists during its continuance. The influence of acute gout in the production of Pneumonia appears to be much less marked than that of rheumatism.

Other febrile states associated with disordered conditions of the blood are frequently causes of Pneumonia; but at present these cases have not been fully analysed with regard to the mechanism of its production. Thus the statistics of Mr. Erichsen[5] show that it is common after severe surgical operations, 45 per cent. of deaths from these causes presenting signs of inflammation of the lungs. It also appears to be common in puerperal fever.[6]

Grisolle states that five-sixths of children affected with gangrene of the mouth suffered from intercurrent Pneumonia. It is also very common in the course of scurvy and purpura. In the latter disease I have seen it assume anatomically the acute primary form.

Albuminuria, associated with disease of the kidneys, is again a very common cause. Rayer found Pneumonia in one-twelfth of these cases. The collection by Jaccoud,[8] of Frerichs and Rosenstein's returns, shows

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[1] See Vol. I. art. Erysipelas, by Dr. Reynolds, p. 687.

[2] See a case by Gubler, quoted in a thesis by Labbé, "De l'Erysipèle," Thèses de Paris, 1858, p. 57. Vulpian, Pneum. Second.

[3] Dr. Fuller, in 268 cases of acute rheumatism, observed 28 of Pneumonia. Dr. Latham, "Dis. of Heart," i. 161, in 136 cases found Pneumonia in 18. Dr. John Taylor (Med.-Chir. Trans. 1845, vol. x. p. 565) only observed it three times in 86 cases. My own observations would lead me to the belief that this complication is not infrequent. I have seen several cases of this class. In the autumn of 1865-6, several cases of acute rheumatism simultaneously admitted into hospital suffered from Pneumonia.

[4] Grisolle, p. 173, cites three cases of this kind; one from Andral, "Clin. Méd.," iii. 463: and a fourth where in two consecutive attacks of acute rheumatism occurring at intervals of some years in the same patient, Pneumonia appeared and disappeared "eight or ten times, following the same course, and having the same duration as the joint affection."

[5] Med.-Chir. Trans. vol. xxvi.

[6] Tonnelé (Arch. Gén. xxii. 487) found Pneumonia in one-twelfth of the fatal cases of puerperal fever. Grisolle, p. 165, says that perimetritis is associated with septic pleurisy, and not with Pneumonia, while uterine phlebitis is more commonly associated with Pneumonia.

[7] Dr. Grainger Stewart (Bright's Diseases of the Kidney) gives the following data of the frequency of Pneumonia in the different diseases of the kidney associated with albuminuria:—acute nephritis, 21 per cent.; contracted or cirrhotic kidney, 7 per cent.; waxy kidney, 4 per cent. [8] Leç. Clin. Méd. 1867.

that the affection was found in 52 of 416 cases, or in 12·8 per cent. Dr. John Taylor,[1] however, found the frequency of Pneumonia to be 24 per cent. Becquerel,[2] in 129 cases of Bright's disease, found Pneumonia in 20 per cent.; but in 100 cases examined by Dr. Bright[3] it was only found in 6 per cent. Rosenstein[4] considers that Pneumonia in this disease is nearly as frequent as pleurisy.

It has been already stated that Pneumonia is a very common secondary result of bronchitis. The relation of the two diseases is threefold. In some cases they appear to be due to a common cause, and acute primary lobar Pneumonia may originate simultaneously with a general bronchitis. In other cases it appears to be the result of a direct extension from the bronchi, and under these circumstances it may appear in the form of "lobular" or "vesicular" Pneumonia; but in other instances the disease thus originating is in the lobar form, and offers no distinctive characters from the primary disease.[5] In a third class the Pneumonia is produced by the intervention of collapse, the mechanism of which process will be further considered hereafter. Bronchitis in the adult, as it would appear from the analysis by Grisolle, often precedes Pneumonia, having been observed by him as an antecedent in 76 out of 201 cases, and in 53 of these the catarrh was recent, *i.e.* it had commenced within a month or three weeks before the Pneumonia appeared. Such antecedent bronchial catarrh, according to Grisolle's experience, is less common in the summer months. My own observations would lead me to the belief that bronchial catarrh may not infrequently precede by two or three days the symptoms of invasion of Pneumonia. Bronchitis commonly precedes the Pneumonia of the aged.[6]

Phthisis is so commonly complicated with Pneumonia, that the latter may, as stated by Dr. Addison, be regarded as the immediate cause of a large proportion of the phenomena of this disease. The question of their mutual relations belongs, however, rather to the subject of Phthisis than to that of Pneumonia.

Congestive conditions of the pulmonary circulation are also a common cause of the disease. The influence of cardiac affections in its production is a very important one, and Pneumonia tends to appear in their course in a proportion of from one-third to one-fifth (Grisolle

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[1] Loc. cit. p. 565.　　　　　　　　[2] Seméiotique des Urines, 1841.

[3] Guy's Hosp. Rep., 1836.

[4] Path. Therap. Nierenkrankheiten, p. 198.

[5] Some authors, and Rilliet and Barthez in particular, consider that this form results from extension of the disseminated variety, and they have termed it, "Pneumonie vésiculaire," or "disséminée," or "lobulaire généralisée." It does not appear to me that this distinction can be always maintained; and, further, it not infrequently happens that in cases of acute pulmonary pneumonia, in addition to the lobar form, disseminated nodules are found in other parts of the lungs.

[6] Dilatation of the bronchi appears to be a very common cause of secondary Pneumonia. Thus Barth (Mém. Soc., obs. iii. 1856) met with Pneumonia in 12 out of 40 cases of bronchiectasis. Biermer, Theorie Anat. der Bronch. Erweiterung (Virch. Arch. xix.), found it in 12 out of 54 cases; and Rapp (Verhand. der Würzb. Med. Gesellsch.), in 21 out of 24 cases. The Pneumonia thus met with is in some cases lobar, in others lobular. It occasionally passes into gangrene.

and Dr. King Chambers).[1] It is probably mainly to the influence of congestion that the inflammatory changes appearing in collapsed portions of lung are due, and it is not impossible that it contributes in a considerable degree to the Pneumonia complicating capillary bronchitis. The influence also of congestion in the production of the inflammation of the lungs which attends the later periods of life, when it mainly occurs in the most dependent parts of the lung—the hypostatic pneumonia of Piorry—is unanimously admitted;[2] Pneumonia being found in a proportion of one-sixth to one-seventh of chronic and cancerous diseases,[3] and of one-fifth of chronic diseases of the nervous system.[4]

ACUTE PRIMARY PNEUMONIA.

SYMPTOMS.—The *invasion* of the disease is sometimes preceded by *prodromata*, which may exist for one or two days, or even longer,[5] before the outbreak of the severer symptoms. They are, however, very frequently wanting:[6] when present they may exist as before stated, as a slight degree of bronchial catarrh, or in the form of general *malaise*, chilliness, loss of appetite, headache, pains and aching in the back and limbs, and an earthy or icteric tint of skin.[7] In old people the disease may be preceded for one or two weeks by headache and vertigo, epistaxis and lumbar pains.[8] Pyrexia of a marked kind is stated to precede sometimes by some days all other signs of the disease, but these cases are exceptional,[9] and it may be questioned whether the pyrexia in such instances has not been caused by a central but undiscovered Pneumonia. There are not, as far as I am aware, any authentic thermo-

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[1] Med.-Chir. Rev., Oct. 1853.

[2] An interesting case of this kind is quoted by Vulpian, "Pneumonies Secondaires," from Rayer, "Mal. des Reins," ii. 293. The patient was obliged to maintain the sitting posture, and the lower portions alone of both lungs were found affected with Pneumonia.

[3] Grisolle.   [4] Calmeil, Dict. de Méd., ii. 196.

[5] Such prodromata may last from one to two weeks, or five or six days, and then the Pneumonia may appear after a slight further exposure to cold. (Grisolle, p. 157.) Zimmermann (Prager Vierteljahresch. 1852, vol. xxxii. p. 97) gives a case in a young man where the prodromata had lasted a week. It may, however, be doubted how far these symptoms can be regarded as being in any respect special forerunners of the inflammation of the lungs; or whether they are not rather to be considered as symptoms of a bad state of health which predisposes to the disease.

[6] Grisolle estimates the frequency with which prodromata are observed in the adult as about one-quarter of all cases. In those to which I have had access the proportion has been 15 out of 53. They are much less commonly observed in children and in old people. Durand-Fardel noted their presumed absence in 20 out of 50 cases (Mal. des Vieillards, 470).

[7] Andral (Clin. Méd. ii. 284, obs. iv.) gives a case of a female who, after drinking largely while heated, was seized with diarrhœa and bronchitis; after ten days the diarrhœa ceased, and signs of Pneumonia then appeared.

[8] Hourmann et Dechambre, Arch. Gén. 2ᵉ. Sér. xii.

[9] Grisolle, p. 187; Traube, Deutsche Klinik, 1857, p. 22.

metric observations recorded of the temperature during the prodromal period,[1] but Huss states that a slight degree of feverishness is sometimes observed. In a large proportion of cases, however, the disease commences suddenly and without previous warning, in persons who up to the moment of seizure had felt perfectly well, and it is not uncommon for the invasion to occur during the night after the patient has gone to bed in his usual health.

The invasion is most commonly marked by rigors, which are generally of a severe character. They form one of the most constant features of Pneumonia in adults, and their frequency and intensity are greater in this than in almost any other disease, with the exception of intermittent fever, pyæmia, and puerperal fever.[2] They are, however, commonly absent in most cases of secondary Pneumonia, and also in that succeeding to long-continued bronchitis. The rigor usually only occurs at the commencement of the disease, and it is rarely repeated, though this is sometimes observed.[3] In some cases it may appear subsequently to other symptoms, such as pain or cough, and in other instances it commences suddenly, after *malaise* and a general feeling of illness have existed during some days. When rigors are absent, the invasion of the disease may be evidenced by other symptoms, such as great prostration and pyrexia. In children also it may be marked by symptoms indicating the early implication of the nervous system, such as convulsions, vomiting, and headache or delirium, which may occur suddenly and without previous warning, or by the milder symptoms of stupor, restlessness, and loss of appetite. In old people sudden prostration and a semi-comatose state may be the first symptoms observed.[4] Rigors may precede by a period of from twelve to twenty-four hours, or even in some cases, of from three to four days, all other symptoms and local signs of Pneumonia with the exception of pyrexia.[5] More commonly, however, other symptoms occur early, and particularly pain in the side, dyspnœa, oppression of the chest, cough, and rusty expectoration. In some cases

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[1] The only case absolutely bearing on this subject with which I am acquainted is one by Monthus, " Essai sur la Pneumonie Double." A patient was in the hospital for abscess of the foot. Her temperature had been normal throughout. One night she got a chill from a draught of cold air. In the morning she felt ill, and the temperature was 100·4 ; within a quarter of an hour a rigor supervened. At the commencement of the rigor the temperature was 100·9. During the rigor, and for an hour after, the temperature was 105·8, it then fell to and remained at 103·6. On the following day crepitation appeared in the lung, and the temperature was 104°.

[2] Huss (loc. cit.) observed rigors in 80 per cent. of his cases. They occurred in 145 out of 182 cases observed by Grisolle, and in 110 of these they were the first symptom noticed. Louis observed rigors as the initial symptom in 61 out of 79 cases. I find their entire absence recorded in 9 only out of 53 cases. In 34 they were distinctly present.

[3] Louis, Rech. Fièvre Typh. ii. 128.

[4] In 35 cases of Pneumonia in old people, Durand-Fardel observed the phenomena of invasion to be as follows :—In 7, rigors only ; in 8, rigors and pain in the side ; in 6, rigors and vomiting ; in 8, pain in the side alone ; and in 6, vomiting alone. Dyspnœa was rare at the outset, and was only observed in 12 out of 50 cases. It was comparatively constant at later periods.

[5] This state, when protracted, constitutes the " Febris Pneumonica " of older writers. The term is also applied to some forms of " Latent Pneumonia."

the earlier symptoms may be headache, or vomiting, or diarrhœa;[1] severe lumbago is also occasionally observed.

Of the symptoms indicating the pulmonary affection, pain in the side is one of the earliest and the most constant, and it may be the first symptom noticed, in some cases preceding the rigor.[2] It is commonly very acute, and its presence is the cause of great anxiety and distress to the patient. It usually corresponds to the site of the Pneumonia, but exceptions to this are occasionally observed, and it assumes at times the character of lumbago. Much discussion has arisen as to its cause, but probably in most cases it is to be attributed to concomitant implication of the pleura. It generally continues during the earlier stages of the disease, tending to diminish towards the third or fourth day, but sometimes lasting until the eighth or ninth. It is aggravated by deep inspirations and by cough, and it occasionally co-exists with marked tenderness on pressure: I have observed it to be associated with considerable cutaneous hyperæsthesia of the affected side.

The other symptoms of the declared disease usually show themselves within twenty-four hours of the invasion, and the aspect of the patient is then to a certain degree characteristic. There is great prostration—a flushed but somewhat earthy or dusky tint of face, tending in some cases to lividity. The skin is pungently hot, sometimes dry, but not unfrequently perspiring. The countenance is expressive of anxiety, particularly when pain is present; at other times the expression is dull and heavy. The respiration is accelerated, and when pain is severe it is shallow and irregular, and the expansion of the *alæ nasi* with the respiratory act is strongly exaggerated. Dyspnœa and a great sense of thoracic oppression are frequently but not constantly present. Speech is rendered difficult and broken by the accelerated respiration, the dyspnœa, the cough, and the thoracic pain. There is a short hacking cough, attended with a laboured expectoration of viscous, tenacious, and rusty sputa. The pulse is accelerated, it is full, and occasionally resisting, but more commonly it is soft, or small, or dichrotous. The decumbency is in most cases dorsal; orthopnœa is less frequently observed. Tremors and subsultus tendinum mark severe cases, which may also be complicated by convulsions or delirium. The urine is scanty and high-coloured. There is complete anorexia and great thirst; the tongue is dry and furred, and the lips cracked; vomiting is sometimes present; the bowels are usually confined, but diarrhœa is by no means rare. These

[1] Headache occurred among the first symptoms in 12, and vomiting in 9, out of 53 cases which I have been able to analyse. For the opportunities of making many of these observations I am indebted to the kindness of my colleagues, Sir W. Jenner, Dr. Hare, and Dr. Reynolds, who have allowed me access to the pulmonary cases under their care, and also have permitted me to use their case-books to supplement, when necessary, my own observations.

[2] In 201 cases analysed by Grisolle, pain in the side was only absent in 29. In 182 it appeared within the first twelve hours in 121. In 4 it only appeared on the third or fourth day, and in 2 of these latter its invasion was marked by the recurrence of an intense rigor.

symptoms may last with unabated or even with increasing intensity, for a period varying from the third to the tenth day of the disease, within which time a notable improvement is usually suddenly observed; the temperature falls abruptly, the flush disappears and gives way to pallor; the skin becomes bathed with a profuse perspiration; the pulse and respiration, particularly the latter, fall in frequency; the dyspnœa and distress are markedly diminished; the cough becomes freer and looser, and the rusty sputa ordinarily disappear. In favourable cases the patient at once feels and declares himself better, and the appetite may return immediately; while in severe cases, or in weakly patients, in spite of the fall of temperature, an intense degree of prostration, amounting even to collapse, and sometimes ending fatally, ensues. When this crisis has taken place there is usually a rapid and continuous improvement both in the general symptoms and in the physical signs, which may, however, be occasionally interrupted by a relapse and by a return of the febrile condition after an interval of twenty-four, forty-eight, or seventy-two hours. In some cases, however, the crisis is indeterminate, the defervescence of the pyrexia is gradual, and the improvement slow and protracted. In unfavourable cases, death may occur from asphyxia or collapse within the first ten days, without the subsidence of the pyrexia; or even, as before stated, after the temperature has fallen to the normal standard.

The symptoms now enumerated require, however, a more special consideration.

Respiratory System.—Accelerated respiration and dyspnœa are among the most marked phenomena of Pneumonia. The latter is not, however, constant as a subjective symptom, and seems to bear, in many cases, no relation to the rapidity of the breathing.[1] It is, however, occasionally the first symptom observed, especially in secondary pneumonias, and it may exist to an intense degree, producing a sense of impending asphyxia; in children it occasionally occurs in suffocative paroxysms threatening death.[2] Its intensity is commonly but not constantly in proportion to the rapidity of the invasion and of the extension of the disease. It is much aggravated by the co-existence of general bronchitis together with the Pneumonia. It has been said to be more intense when the inflammation affects the apex of the lung, but Grisolle has shown that facts do not confirm this opinion. In some cases the sensation of dyspnœa is probably masked by the prostration of the nervous system.

The rate of respiration is greatly quickened. The number of respirations per minute is seldom less than 30, often 35 to 40, and they may even reach 60 or 70. The acceleration of the breathing is

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[1] Dr. Walshe says that he has seen patients breathing at the rate of 50 or 60 per minute without any sense of dyspnœa. It is possible that in some of these cases the blood is sufficiently aërated by the accelerated breathing to prevent the sense of dyspnœa being felt. In some cases, when the respiration is less rapid, the sense of dyspnœa is extreme.
[2] Ziemssen, loc. cit.

generally proportionate to the extent of lung affected, but this is not invariably the case. It is increased by co-existing bronchitis, or by any cause interfering with the thoracic expansion, such as pregnancy. The acceleration of the breathing is proportionately greater than that of the pulse, and hence arises the perverted pulse-respiration-ratio which is especially insisted on by Dr. Walshe as one of the earliest signs of Pneumonia.[1] This perversion may reach the limits of 60 respirations to 100 pulsations per minute ; or in some cases, when the pulse remains slow, the ratio has been observed of 56 pulsations to 60 or 70 respirations per minute.[2] The respiration is commonly more rapid in children than in adults, and in them the anhelation may be extreme and the respiratory movements irregular. In some cases of asthenic Pneumonia the pulse-respiration-ratio may not vary markedly from that of health.[3] It is not improbable that the extreme degrees of frequency of respiration may in some cases be due indirectly to peculiar states of the nervous system.[4] The breathing, in addition to being rapid, is commonly shallow, particularly when pain in the side is severe.

Cough is not only an almost constant, but it is one of the earliest symptoms.[5] It is short and hacking, and rarely paroxysmal, though it sometimes becomes so in children in the later stages of the disease. The violent paroxysms, resembling those of hooping-cough, mentioned by Rilliet and Barthez, are thought by Ziemssen to be more characteristic of Broncho-pneumonia. It is often, on the other hand, less frequent in old people and in children than in adults, and it has been observed in the former, that a cough previously existing, and caused by bronchitis, has ceased or has become greatly diminished on the invasion of Pneumonia. The cough often ceases when a fatal termination is approaching.

The expectoration which attends the cough usually presents characteristic features, depending on the admixture of blood. This is not, however, always apparent at the outset, when the sputa may be frothy and aërated. They soon, however, tend to become peculiarly viscous, adhering with great tenacity to the containing vessel, and owing to this quality they are often expectorated with great difficulty. They are at the same time transparent, having various tinges of reddish brown or saffron, or they may be of a lighter tint, resembling apricot jelly or barley-sugar. The most common colour is that characterised familiarly as "rusty," which aptly expresses their appearance. In rarer cases they may sometimes, in the earlier stages, present a brighter tint, or even a rose colour, but this is by no means so strongly marked as in the earlier stages of acute bronchitis, though in Pneumonia the

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[1] Dr. Walshe (Dis. of Lungs, Ed. 1860, p. 366) says that the return to the normal ratio may, on the other hand, be one of the first signs of improvement.
[2] Ibid.   [3] Ibid.
[4] Traube (Annalen der Charité, vol. i.) considers that the rapidity of the breathing is in part due to pain, and in part to the high temperature of the blood affecting the nervous centres, as it is diminished by the application of cold.
[5] In 8-9ths of cases (Grisolle).

sputa of the first few hours are commonly of a brighter red than subsequently, and streaks and specks of blood may appear in them. Dr. Walshe remarks that profuse hæmoptysis is commonly a sign of co-existing tuberculosis. In the cases when I have seen this, the same connexion has been distinct. Huss also confirms this observation, but adds that in the Pneumonia complicating heart disease, the sputa may contain an unusual quantity of blood.

In other cases the sputa may be more watery, almost diffluent, of a dark purple colour, and occasionally offensive. This appearance, familiarly known as "prune juice," and which probably results, in part at least, from the presence of œdema of the lungs, is commonly considered a sign of grey hepatization; but this connexion is by no means invariable, for such sputa may co-exist with red hepatization,[1] and may be absent when grey hepatization is found *post mortem*; [2] sputa of this character are, however, to be regarded as indicative of a grave form of the disease.

The sputa which have now been described may be regarded as almost pathognomonic of the pneumonic process in some of its forms, but variations in their characters may be sometimes observed, and they are said at times to present a greenish tint.[3] They may also at times exhibit appearances during some days, differing but little from those of bronchitic sputa, and without any blood-tinge which is appreciable to the naked eye, but in these cases they are commonly more tenacious and gelatinous than those seen in simple bronchitis.[4] In severe cases they may be simply purulent throughout.[5] In other instances the viscidity is less apparent, and the prune-juice sputa in particular are often diffluent and watery.

The rusty sputa are, however, the most usual form, and they commonly are present early in the disease, and are among its first symptoms.[6] The time of their appearance is, however, often considerably

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[1] Cases are recorded by Andral where prune-juice sputa preceded all the physical signs of consolidation (Clin. Méd. iii. obs. 28, p. 361), and when they co-existed only with red hepatization (Ib. obs. 39, p. 392); also another case where they appeared on the fourth day, but were replaced on the subsequent day by ordinary expectoration, the patient proceeding afterwards to recovery (Ib. obs. 40, p. 393). Durand-Fardel has also noticed them in the earlier periods of the disease (Mal. des Vieillards, 477). Huss also states that prune-juice sputa are by no means constant accompaniments of grey hepatization, but that they are common in the Pneumonia of drunkards, and also in tubercular Pneumonia.

[2] See Andral, loc. cit. obs. 24, pp. 350-1. Of two fatal cases of grey hepatization coming under my own observation, this character was not observed in one. In the other the sputa were diffluent, and had the tint of burnt sienna.

[3] Grisolle. These have not come under my own observation in the earlier stages. A greenish tint is not uncommon during the period of resolution. When icterus complicates Pneumonia, a greenish tint is sometimes observed. (Andral, loc. cit. obs. 55, p. 440.)

[4] Sputa of this kind are, however, more common in Pneumonia which is secondary to bronchitis, and particularly in the Pneumonia accompanying influenza.

[5] Only one such case has come under my own observation.

[6] In 191 cases observed by Grisolle, characteristic sputa existed on the second day of the disease in 71; they were present in 33 of 53 cases analysed by myself. Of the cases in which they were absent, 4 were in children, in another the Pneumonia was secondary to albuminuria, and they were absent in one fatal case.

delayed, and they may not be seen until the fifth or sixth day, or even until the twelfth day. In one case coming under my own observation, there was not a vestige of expectoration until the tenth day, when the Pneumonia was rapidly approaching resolution, and the amount was then limited to two small rusty masses expectorated on each of two consecutive days.[1] When present, they commonly continue through the first five or six days, but they may preserve their rusty tint until the ninth day.[2]

In some cases, otherwise typical, no expectoration whatever occurs throughout the whole course of the disease. In others the characteristic sputa may be absent during the acute period, and only a moderate amount of bronchitic, or slightly purulent, or pigmented sputa may appear during resolution. In cases of grey hepatization and of abscess of the lung the sputa may be purulent or creamy-looking. When gangrene supervenes they become offensive, and fragments and débris of pulmonary tissue may be found in them. The entire absence of expectoration is said to be more common in Pneumonia of the apex than in that of the base of the lung.[3] Children under six years of age seldom if ever expectorate, but Ziemssen says that he has found the rusty tint in the sputa of infants when vomiting has taken place. They are also often absent or only mucoid in the Pneumonia of old age,[4] and in many cases of secondary intercurrent Pneumonia, and in that complicating delirium tremens. The sputa often cease or fail to be expectorated when the disease is approaching a fatal termination.[5]

Both purgation and bleeding diminish or check the expectoration.

When Pneumonia complicates other diseases of the lungs, the rusty sputa may be more or less masked by other forms of expectoration present, or they may replace these.

Remak[6] first described, as one of the phenomena of Pneumonia, casts of the air-vesicles and of the minuter bronchial tubes, which may be found in the sputa when these are floated in water. He

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[1] Grisolle questions whether rusty sputa expectorated only during convalescence may not be considered critical. In the case in question they only occurred after the thermometric crisis, and their amount is almost invariably too small to permit them to be regarded as a true critical evacuation.

[2] Exceptional cases are recorded where viscous and rusty sputa may continue during longer periods, as in a case by Dr. Stokes, "Diseases of Chest," 361, where a patient with broken ribs continued to expectorate sputa of this character for weeks after the physical signs of Pneumonia had disappeared. Andral also quotes a case where rusty sputa continued to the nineteenth day, lasting nine days after all physical signs had disappeared. (Loc. cit. 526.)

[3] Out of 14 cases where the sputa were entirely absent, in 7 the apex was the site. (Grisolle.)

[4] Rusty sputa were observed in 17 out of 61 cases of Pneumonia in old people observed by Hourmann and Dechambre, and in 18 out of 50 cases observed by Durand-Fardel.

[5] The absence of expectoration may, as suggested by Andral, be sometimes due to the absence of bronchitis, or to the viscosity of the exudation in the air-vessels. The absence of expectoration in some cases where resolution is very rapid is a remarkable evidence of the absorptive power of the lung.

[6] Diagnostische und Pathognostische Untersuchungen, 1845.

regarded them as pathognomonic of the exudative period, but they are by no means constant.[1]

Histologically the main elements of pneumonic sputa consist of swollen epithelium cells, which have assumed, by imbibition, the spheroidal form ; large mucoid cells, sometimes with double nuclei, and occasionally. tinged with imbibed hæmatine ; swollen cells of columnar epithelium, occasional granule cells, free oil-globules and blood-discs. Dr. Walshe says that true pus cells are never found in the rusty sputa of Pneumonia. They may, however, appear when the disease is approaching resolution, and in this stage large round cells containing granules of black pigment become a very predominant feature, mingled with free nuclei, free pigment-granules, and much granular débris. Chemically the sputa contain mucus and albumen.[2] Sugar has been observed in them by Dr. Walshe[3] and by Dr. Beale,[4] and tyrosine by Griesinger.[5] They contain, in the earlier stages, a small amount of organic constituents, and an excess of fixed salts[6] in proportion to the serum of the blood, but this excess is reduced during resolution, when the fixed salts are diminished in amount, probably owing to their elimination by the kidneys. Among these salts the chlorides are sometimes in excess.

In some cases the sputa tend to assume an acid reaction. This was noticed by Dr. Beale, who suggested that it might be due to the pneumic acid discovered by Verdeil, and found by him to be increased in the inflamed lung.[7] The true explanation would appear to be that afforded by the observations of Bamberger, that they are markedly deficient in alkaline phosphates when contrasted with the sputa of simple catarrh.[8]

The expired air, as Nysten and Dr. Walshe have observed, is colder

[1] Biermer says that, in 25 cases, he failed to find them six times. (Die Lehre von Answurf, p. 52.)

[2] Scherer, quoted by Biermer, loc. cit. p. 114.   [3] Loc. cit. p. 367.

[4] Med.-Chir. Trans. vol. xxxv.

[5] Bleuler, Clin. Beobach. ueber Pneumonie, Diss. Inaug. (Zurich, 1865), p. 37. There was no icterus in this case. The patient recovered.

[6] Biermer, loc. cit. Beale, loc. cit. In three cases Dr. Beale found the following proportions of fixed salts and of chloride of sodium in the sputa :—

In 100 parts of solid matter.	1st case.	2nd case.	3rd case.
Fixed salts	24·78	32·86	20·67
Chloride of sodium	10·12	18·11	12·67

In another fatal case where the sputa contained 9·83 per cent. of fixed salts in the solid matter, the blood taken from the heart contained only 2·82 per cent. Healthy pulmonary mucus, according to Hasse, may contain 18 per cent. of fixed salts, and the mucus of influenza, according to Wright, contains 8·9 per cent. (Quoted by Dr. Beale.)

[7] See Gaz. Méd. 1851, p. 777 ; also Robin et Verdeil, Chem. Anat. Phys. ii. 460-1.

[8] Wurzburg Med. Zeitsch. ii. Nos. 5 and 6. Bamberger's observations contain the following interesting facts in relation to the composition of pneumonic sputa :—

(a) They contain no alkaline phosphates, while catarrhal sputa contain 10 to 14 per cent. of alkaline earths.

(b) In catarrh the soda is to the potash as 31 to 20, while in Pneumonia the soda is to the potash as 15 to 41.

(c) Sulphuric acid in catarrh is equal to 3 per cent., in Pneumonia to 8 per cent.

(d) At the period of resolution the chemical character of pneumonic sputa approaches the catarrhal type.

than natural, and the amount of carbonic acid excreted is also diminished.[1]

The physical signs indicative of the disease commonly make their appearance within twenty-four or forty-eight hours from the symptoms of invasion, but they may be undiscoverable for three or even four days, though probably when their appearance is thus delayed the Pneumonia may be central.

In the order of their typical sequence they may be stated to consist of the following signs, corresponding to the anatomical stages of engorgement, hepatization, and resolution: among them, however, certain varieties occur :—

(1) Altered characters of the respiratory sound, which may be either weaker or harsher than natural, and attended or immediately followed by fine, crackling râles.

(2) Dulness on percussion, attended by bronchial or tubular or suppressed breathing, bronchophony, and increased vocal fremitus, together with diminished respiratory movement, chiefly affecting the act of expansion.

(3) The return of crepitation, usually in a coarser form ;—gradual diminution of percussion dulness, together with the return of the respiratory movements and of the characters of the respiration and of the vocal resonance and fremitus to the healthy standard.

(1) *The Congestive Stage.*—The indications of this stage are in most cases somewhat uncertain. There may, however, occasionally be noticed, even at an early period, a deficiency of pulmonary tone on percussion, not amounting to absolute dulness, but presenting this character in an increasing degree as hepatization advances. In some cases, however, during the early stages, the percussion note may be distinctly tympanitic. Sometimes, as was first noticed by Dr. Stokes, an increased harshness of the respiratory murmur may be the first phenomenon observed, but this is not constantly present, although it may occasionally be heard at the confines of a part where hepatization is extending. In some cases, however, the respiratory murmur is weakened and loses in clearness and softness, acquiring almost *ab initio* the character of the "indeterminate" breathing of Skoda.[2]

The existence of fine crepitation in this stage is less constant ; when present its characters may be best described in the terms of Dr. Walshe as occurring "in puffs more or less prolonged, but rapidly evolved, composed of a variable, sometimes immense number of sharp crackling sounds, all perfectly similar to each other ; conveying the notion of minute size ; dry ; co-existing exclusively, except in rare cases, with inspiration ; and, once established, remaining a persistent

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[1] Walshe, loc. cit. This latter phenomenon is common to many acute diseases.

[2] Dr. Walshe, p. 355, states that when congestion is near the surface the respiration is weaker and harsher. When the part affected is deeply seated, the intervening healthy pulmonary tissue may give rise to puerile breathing. He has also observed a "fair number of cases in which exaggerated breathing, coupled with febrile excitement and slight pain in the side, were the earliest indications of a central Pneumonia eventually travelling to the surface."

condition until superseded by other phenomena." The simile intro-
duced by Dr. Williams, between this râle and the sound produced by
rubbing the hair between the fingers close to the ear, is so truthful as
to have become almost proverbial. For its proper evolution it is often
necessary that a full inspiration should be taken. It requires, under
these circumstances, to be distinguished from the râle produced on
the first full expansion of a portion of the lung which has been pre-
viously in a condition of imperfect action, either from muscular weak-
ness or from pleurodynia. The latter, however, disappears after one
or two deep respirations, while the true crepitant râle is, as above
stated, persistent.

The crepitant râle may often be mingled with sibilant or sonorous
râles; or, in other cases, where pre-existing bronchitis passes into
Pneumonia, with coarser bubbling râles.

The râle is often wanting in children, and both in them and in old
people it is commonly coarser and less rapidly evolved than in the
Pneumonia of adults.

In some cases, however, when the stage of engorgement passes
rapidly into that of hepatization, crepitation is not heard, even
though the Pneumonia be developed under direct observation.[1] Occa-
sionally it may only be heard after bronchial breathing has appeared.

The mechanism of the crepitant râle is not yet determined. The
two leading theories respecting its mode of production are (1) that it
may be produced by air and the viscous exudation matter in the pul-
monary vesicles, and (2) that it is due to the expansion of the parietes
of the vesicles previously agglutinated together.[2]

In rare cases bronchial breathing may be heard during the con-
gestive stage.[3] Vocal fremitus and vocal resonance are increased in
proportion to the condensation of the pulmonary tissue, but true
bronchophony is not heard.

(2) *Stage of Hepatization.*—The crepitant râle last described may
disappear at various periods of the second stage, or it may continue
throughout its entire course, becoming coarser as resolution advances.

The characteristic physical signs of the second stage depend, how-
ever, on the filling of the air-vesicles with the products of inflamma-
tion, by which the part so affected is distended to the degree of
medium or full insufflation. In consequence of this all further ex-
pansion movement of the affected part ceases, though thoracic eleva-
tion continues, and a certain though not extensive degree of enlarge-

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[1] Walshe, loc. cit. 356. Dr. Walshe states also that "the diagnosis of Pneumonia must
be made once in every four or five cases independently of the crepitant rhonchus" (loc.
cit. 337). In the writings of Laennec and Andral great stress is laid on this râle. The
latter author describes, on the strength of its persistence, a case where the stage of engorge-
ment lasted eight days, and ended fatally without hepatization. It is probable that this
was only a case of capillary bronchitis. (Clin. Méd. iii. 297, obs. viii.)

[2] Walshe. Dr. Walshe, however, has observed a râle indistinguishable from true
pneumonic crepitus in some cases of pulmonary œdema. (Loc. cit. p. 123.)

[3] Traube (Annalen der Charité, i. 286) says that bronchial breathing may occur during
the stage of engorgement when the combined effect of œdema and of swelling of the
pulmonary tissue is sufficiently great to expel all air from the pulmonary vesicles.

ment of the affected side may ensue.[1] The distended lung may even
encroach on the mediastinum,[2] and may occasionally cause a slight
displacement of the heart.[3] The prominence or obliteration of the
intercostal depressions is, however, not seen to the extent observed
in cases of pleuritic effusion, although they sink to a less degree than
normal during the act of inspiration.

The percussion note over the affected part loses its normal pulmo-
nary resonance. In some cases it becomes in the early stages slightly
tympanitic in quality, and it may retain this character anteriorly
when the dull note posteriorly indicates complete consolidation of
the latter region. Over lung completely consolidated, the percussion
note may be almost toneless, and the sense of resistance is greatly
increased, though neither of these qualities are so strongly marked as
in the presence of extensive pleuritic effusion.

Instead of absolute tonelessness the note may, however, be tubular
or amphoric. The tympanitic quality is less common when the
consolidation has attained its maximum intensity,[4] but it sometimes
returns during the progress of resolution. In the earlier stages it is
often necessary to compare the percussion note on the two sides, in
order to detect a slight degree of dulness on that affected. The con-
trast becomes increasingly marked as consolidation advances.

When the Pneumonia affects the base, the upper part of the lung
often continues to give excessive or even tympanitic resonance ; and a
cracked-pot sound may sometimes be elicited here when the chest
wall is elastic. The note under the clavicle is, however, rarely so
markedly amphoric or tubular as that found in the same situation in
cases of pleuritic effusion.[5] The limits of percussion dulness are
sometimes sharply defined, but occasionally they are indistinct. In
the latter case the percussion note at the margins of the inflamed
part may yield a tympanitic resonance, or may have its natural
resonance impaired by a pneumonia extending deeply.

Blowing or bronchial respiration passing into tubular and intensely
metallic breathing, distinguish this stage.[6] These characters usually
succeed those of the first period with great rapidity. Grisolle de-
scribes, under the title of " bruit de taffetas," from its resemblance
to the noise produced by the tearing of linen, an intermediate sound,
occasionally heard between the disappearance of the crepitation and

[1] This point has been the subject of considerable discussion, but the enlargement
appears to be settled in the affirmative. Dr. Walshe, however, states that general
enlargement of the side is never the resultant of Pneumonia alone.

[2] Walshe.   [3] Ibid.

[4] This is noticed by Skoda, and referred by him to the lung still containing some air—
a proposition also maintained by Dr. Hayden (Dublin Journ. 1866, xli.) Dr. Bäumler
attributes it in some cases to relaxation of pulmonary tissue, in others to a note conducted
from the larger bronchi. (Deutsch. Arch. Klin. Med. i. 145.)

[5] Walshe.

[6] "The tubular form (of respiration) occurs in perfection in but one condition of lung,
that of hepatization ; so true is this, that tubular and pneumonic breathing may be used
as convertible phrases, but not infrequently Pneumonia runs its course without having
produced true tubular breathing, diffused blowing alone being audible." (Walshe,
loc. cit. 112.)

the supervention of bronchial breathing. The bronchial character is heard first during expiration, but it subsequently attends the inspiratory sound also.

In some cases, however, when there is no evidence of pleuritic effusion, all respiratory sound may be completely absent over hepatized lung. The cause of this is uncertain, for post-mortem evidence has shown that it does not necessarily depend on complete exclusion of air from a large tract of lung, since under such circumstances tubular and bronchial breathing may persist, and, on the other hand, respiration may be absent when only a small portion of pulmonary tissue is affected; nor does it necessarily depend on the obstruction of the bronchi by exudation matter.[1] In other cases tubular breathing may alternate with absence of respiration.[2] The intensity of the bronchial or tubular breathing appears to depend in some measure on the size of the bronchial tubes included within the portion of lung affected.

The vocal resonance is increased in intensity, and is at the same time altered in quality, acquiring the character known as bronchophony. The cough may also acquire a bronchial character. Intense whispering pectoriloquy may be occasionally heard.[3] The heart's sounds are also sometimes heard with undue intensity over the affected lung.[4]

The vocal fremitus is generally increased over the affected side. In comparatively rare cases, however, this is not observed; the difference between the two sides may be so slight as to be scarcely perceptible, or the fremitus may even be less on the affected side. In some exceptional cases, however, vocal fremitus, vocal resonance, and the respiratory murmur may all be simultaneously absent,—a condition when the diagnosis from a case of pleurisy might present some difficulties.[5] (See Diagnosis.)

In some instances pulsation may be felt over the affected lung. It is a disputed point whether this is due to the transmission of the cardiac impulse, or to increased pulsation in the arteries of the inflamed lung.[6]

The signs now enumerated are most distinct when the inflammatory consolidation has reached the surface. When it is seated in the deeper portions of the lung, and the more superficial layers are left unaffected, the physical signs may be comparatively obscure. Laennec thought that crepitation and bronchial breathing could be heard

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[1] This view is however affirmed by Skoda, who says that the auscultatory phenomena of respiration may be restored after coughing.

[2] Dr. Walshe (loc. cit. 360) has traced this in one case to pressure on the main bronchus. Other theories advanced have been that of Grisolle, that it may be due to complete loss of elasticity of the lung; or of Dr. Gairdner, that it is due to collapse from obstruction of the bronchi.

[3] Walshe. This, according to my own observation, is not very uncommon.

[4] Ibid.

[5] Wintrich, Virchow's Handb. vol. v. Abth. i. p. 299. In this case also the bronchi were obstructed by firm exudation matter. The case was mistaken for pleurisy, and paracentesis was attempted. There was no fluid in the pleura.

[6] The latter opinion is denied by Grisolle, but supported by Graves, Stokes, and Skoda, and admitted as a possibility by Dr. Walshe.

deeply; and this may sometimes be the case, though instances occur where neither of these signs are distinct. The signs also derived from alterations in the vocal fremitus, and resonance, are usually wanting under these circumstances.[1] Where inflammation of the pleura complicates the Pneumonia, friction is commonly heard during its whole course. It may, however, be absent during complete consolidation, owing to entire loss of movement of the hepatized lung, and also when effusion is extensive. When this takes place, the dulness increases in extent, and the resistance is greater. Bulging of the affected side becomes more distinct, and displacement of the heart occurs if the left side be affected; the sounds of respiration usually become weaker, and the bronchial breathing less distinct; but the intensity of this, and the site in which it is heard, depend on the proportion of fluid present. The fremitus is commonly diminished. Bronchophony may also be diminished below the fluid, or may continue at its level, or the vocal resonance may in the latter position assume an ægophonic tone.

The period necessary for the evolution of the different physical signs varies. The duration of the initial stage of congestion may, as has been already stated, extend over two or three days, and bronchial breathing and distinct percussion dulness may not appear until the second or even the fourth day, and this appears to be more commonly the case with Pneumonia of the apex. In other cases hepatization may advance so rapidly that a large tract of lung may be consolidated in from twenty-four to forty-eight hours, or bronchial breathing may be heard within twelve hours from the period of invasion.

The condition of the unaffected lung is usually that of increased functional activity. In some cases it is hyper-resonant on percussion, and the respiratory murmur over it, and over the sound parts of the affected side, is of an exaggerated or puerile type. In three cases I have observed that bronchial breathing, friction, and moist râles were heard over the healthy side where resonance on percussion has been perfect, for a distance of more than a hand's breadth extending outwards from the scapula.[2] These signs disappeared *pari passu* with the return of the affected side to a normal condition. The vocal fremitus was not increased over the unaffected side, although a bronchophonic tone of the voice was conducted for a short distance, but not so far as the bronchial breathing. It seems difficult to explain these phenomena on the theory of consonance, and my own conviction is that they are due to direct conduction.

(3) *During the stage of resolution* the abnormal physical signs commonly disappear in an inverse order to that in which they originated. Improvement is generally first manifested by a reappearance of the crepitant râle. This râle—the *rhonchus crepitans redux*—is usually coarser and less rapidly evolved than that heard during the

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[1] This subject will be further alluded to under the head of "Diagnosis."

[2] Barthez and Rilliet (i. 460) describe bronchial breathing as being sometimes heard close to the spine on the unaffected side.

progress of hepatization; it tends to pass into a more liquid form—the subcrepitant râle—and occasionally it acquires a distinctly fine bubbling character. In some instances, however, resolution may proceed rapidly without the occurrence of redux crepitation. Sibilant and sonorous râles also appear in the affected part, and sometimes in other portions of the lung. The dulness on percussion gradually disappears; the tubular breathing diminishes in intensity, it loses its metallic quality, and both it and the bronchial breathing pass into blowing respiration, which finally becomes indeterminate or simply weak. Similar changes occur in the bronchophonic tone of the voice, but the vocal fremitus and resonance usually continue intensified as long as the percussion note remains less resonant than natural.

The signs which persist the longest are some dulness on percussion, and the subcrepitant or fine moist râle, and the latter may often remain during a prolonged period after the other physical signs have disappeared. In some cases, however, when the resolution is very rapid, the redux crepitation may be wanting, and the dulness and altered characters of the respiration may vanish within twenty-four hours, giving place to a weakened or indeterminate respiratory sound. Friction also may continue long after the other physical signs have disappeared.

Generally, though occasional exceptions are observed, the parts last affected are those in which the signs of resolution first appear. In some instances, however, I have noticed the dulness disappear in irregular patches over the consolidated part. When a whole lung has been consolidated, the resolution usually commences at the apex. If this is not the case, the existence of tubercle may be suspected.[1] In cases of double Pneumonia the lung last affected may first show signs of improvement; but occasionally the resolution of that first attacked may progress, while hepatization is still advancing in the other.

(4) The physical signs *of grey hepatization* and of *diffuse suppuration of the lung*,[2] present nothing characteristic. Occasionally a high-pitched metallic bubbling râle, as described by Stokes, supervenes, while dulness on percussion still persists; but I have observed this in a case where, *post mortem*, the lung was found to be almost entirely in a state of red hepatization. The formation of a circumscribed abscess (a very rare event in Pneumonia not arising from secondary deposits in pyohæmia) is only discoverable by the local signs of the formation of a cavity, together with profuse purulent expectoration, which is often offensive. and in which the elastic tissue of the lung may sometimes be found. Gangrene of the lung—also a rare event in primary Pneumonia—is mainly to be recognised by the signs of a cavity, coupled with the peculiar fœtor of the sputa and the expectoration of débris of pulmonary tissue.

[1] Walshe, loc. cit. 372.
[2] It may be doubted whether the latter ought to be described as a separate condition. (See Pathology.)

Circulatory System.—The pulse is almost invariably accelerated, though exceptional cases occasionally occur, particularly in old people,[1] when this is not observed. In adults, in cases of moderate severity it usually ranges from 90 to 120[2] pulsations per minute, but it may reach 130 or 140, and in children 160, 180, or 200, or it may be so rapid as to be uncountable. The extreme degrees of frequency of the pulse in children are commonly only observed in the earlier periods of life. In some cases the pulse may become notably retarded before the fatal issue.[3] A pulse above 130, or even 120, is, except in children, a very unfavourable sign.[4] The frequency of the pulse commonly, but not always, bears a certain proportion to the acceleration of the respiration, and a similar proportion may within certain limits be observed between the frequency of the pulse and the degree of temperature attained.[5]

In characters a pulse of moderate frequency is commonly during the earlier periods full, but soft; it may, however, be tense and incompressible.[6] These characters tend, however, to diminish by the fifth day, when the pulse usually becomes smaller and often acquires a dichrotic character. A rapid pulse is, however, generally both small and weak. A small pulse may at times be associated with signs of distension of the right ventricle, particularly when the Pneumonia is extensive and other signs of defective aëration of the blood are distinct, but it may occasionally be observed when these are not marked, and when the distension of the right side of the heart is not demonstrable either by percussion or by increased post-sternal or epigastric impulse. It is, however, *à priori,* extremely probable, and it is also confirmed by postmortem observation, that overloading of the right cavities of the heart is the direct result of the obstructed pulmonary circulation, and the immediate effect of this will be that a proportionately diminished amount of blood is propelled by the left ventricle into the systemic arteries, though the general injurious effect on the aëration of this fluid is partly compensated for by the increased rapidity of the circulation.

The diagnosis may in some cases be aided by the palpation and auscultation of the heart. If the cardiac impulse be strong and the sounds full when the pulse is small, the over-distension of the right ventricle is probably present. In other instances the impulse is

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[1] Walshe, loc. cit.

[2] In a quarter of Grisolle's cases it was, however, below 100.

[3] Thus in a case by Grisolle, in an old man, the pulse was only 58 for twenty-four hours before death.

[4] Out of 184 cases of recovery, Griesinger observed a pulse of 120 to 150 in fifty-four patients above the age of 15. Twenty-seven patients, having a pulse of this frequency, died. Hence nearly one-third of all the patients above 15 under Griesinger's care, with a pulse above 120, died. The dangerous significance of this symptom rises to an extreme with advancing age. (Bleuler, loc. cit.)

[5] Ziemssen, loc. cit. 217. Griesinger, loc. cit.

[6] It may seem superfluous to point out the fallacy which may arise from rigidity of the arteries from calcification in advanced life, but this condition requires constantly to be remembered in estimating the "strength" of the pulse in acute disease.

weak and the sounds less distinct than natural, and the enfeebled pulse must then be attributed to impaired cardiac power.[1]

A small pulse may therefore be attributed in many cases to the first-named cause, though enfeebled cardiac innervation has probably in some instances a considerable share in its production. A dichrotous pulse must, however, depend in a greater degree on weakened cardiac power and also on diminished tonicity through impaired innervation of the muscular coat of the arteries. The dichrotous character is often extremely marked about the period of the crisis.[2]

The heart's action is commonly more accelerated in weakly people, and also by co-existing cardiac disease, so that a rapid pulse in Pneumonia may occasionally draw attention to this previously unsuspected complication.[3]

Intermittence of the pulse is sometimes observed in adults; it is much more common in the Pneumonia of old age, independently of any discoverable cardiac disease. In children the pulse, when very rapid, is frequently unequal, but not distinctly intermitting.

Occasionally, as remarked by Dr. Graves, a murmur may be heard over the heart during the height of the disease, and may disappear during the progress of resolution. I have also observed this in one case ; the murmur was systolic, and was limited to the apex. In Dr. Graves's case it was heard over a large extent of the affected side. No satisfactory explanation has been offered of this phenomenon. It is difficult to attribute it to polypoid concretions of fibrine, seeing that its disappearance was not attended by any of the phenomena of embolism.

Evidences of impeded circulation through the lung are also observed in the cyanotic tint of the lips, and less commonly of the fingers (though this is sometimes seen in children), and also in the occasional distension of the jugular veins,[4] which may sometimes pulsate ; a similar pulsation has been seen to extend to more distant parts of the venous system.[5]

Epistaxis is sometimes observed. It may be one of the earliest symptoms, or it may appear among the phenomena of the crisis. I have observed it under both sets of circumstances, but not so frequently as has been noticed by some authors.

The condition of the blood will be described under the pathology of the disease.

[1] See some excellent critical remarks on this subject in M. Jaccoud's Clin. Méd. The test proposed by M. Jaccoud of "radial recurrence," *i.e.* of the blood finding its way back by the collateral circulation to the radial artery when compressed superiorly, is, I believe, fallacious as an evidence of cardiac power. It may be observed in the weak and dichrotous pulses of advanced phthisis.

[2] For sphygmographic tracings of different varieties of the pulse in Pneumonia see Appendix A.

[3] Traube, Symptomen der Krankheiten der Respirations-Organe, p. 31.

[4] Grisolle (loc. cit. 257) thinks that this may be occasionally due to pressure by the hepatized lung. It has been observed by him on the affected side in Pneumonia of the apex, and he cites Bouilland as having similarly seen distension of these veins limited to the affected side.

[5] Stokes (loc. cit. 331), quoting from Graves, says that the pulsation was seen in the back of the *hand*. Graves (Clin. Méd. ii. 41) says *head*. (Query a misprint.)

Digestive System.—This also participates in the general pyrexial state. Thirst is marked, and the appetite is lost. The tongue varies in appearance; sometimes it shows but little alteration, but usually it is coated with a thick creamy fur. In severe cases it tends to become dry and brown, and sordes form on the teeth, and the lips are dry and cracked. Difficulty of deglutition is occasionally observed in old people.[1]

Vomiting has been already stated to be an occasional symptom of the invasion. I find this recorded in eight out of fifty-three cases, most of whom were adults; it is much more common in children, occurring, according to Barthez and Rilliet, in one-half, and according to Ziemssen in three-eighths, of all cases of Pneumonia. It usually ceases after the first or second day, but it may sometimes continue throughout the pyrexial period, and even subsequently.[2] I have known a case in which erysipelas followed Pneumonia, and where vomiting continued during three weeks, and placed the patient's life in considerable danger.

Diarrhœa is also an occasional symptom of the invasion, though not so frequently so as vomiting, with which, however, it may co-exist. It rarely continues unchecked throughout the case, except in very young children; it sometimes appears at the period of the crisis or during resolution.[3]

When the gastric symptoms are severe, they have given rise to the description as a special variety of a *gastric* or *bilious form of Pneumonia* (in which, however, the complication with icterus is not included). This variety, which was first described by Stoll, has been the subject of much discussion, and it appears to be a very ill-defined one. A certain number of cases indeed occur in which the symptoms of gastro-duodenal, or enteric catarrh, are very distinct. I have met with three or four such; but all gradations can be observed between these and the more ordinary symptoms evincing participation of the digestive tract in the disturbance occasioned by acute pyrexia.

When the condition is a marked one, the complexion is more opaque and earthy than usual. There is greater prostration, and often the headache is more than commonly severe. The tongue is much loaded, nausea is present, or vomiting may persist throughout. The epigastric region is sometimes tender. Constipation is present in some cases, diarrhœa in others, and the latter often appears towards the crisis. Huss found this form of complication most frequent in the summer months.[4] The ordinary state included under this term does not

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[1] Wunderlich, Abth. iii. B. ii. 363.

[2] Louis (Fièvre Typh. ii. 465) records gastric symptoms, pain or vomiting, in 17 out of 24 cases of Pneumonia which died, and in 23 out of 58 which recovered. In many, these symptoms occurred late in the disease, and it may be questioned whether they were not in part due to antimonial treatment.

[3] Diarrhœa appears to have been very common in Louis' cases, amounting to one-third.

[4] Huss found gastro-intestinal catarrh in 5 per cent., "acute enteritis" in a little more than 1 per cent.

appear to exercise much influence on the mortality of the disease, though cases presenting its more decided features are usually protracted in their course.

Nervous System.—Headache has been already spoken of as an almost constant symptom. It may be very severe, and in such cases it is greatly aggravated by the cough. It usually, however, tends to diminish after the first three or four days. Delirium is also common,[1] but except in patients of dissipated habits, in whom it may assume the characters of delirium tremens, it is rarely violent. It may however occasionally appear in so sudden and severe a form as to be mistaken for acute mania (Grisolle), but most commonly it exists only as a calm wandering, or as an incoherent talkativeness. Huss remarks, contrary to some previous statements on this subject, that delirium is not especially common in Pneumonia of the upper lobes, but that it is most liable to occur when a large tract of lung is affected —either in the single or double form. Under these circumstances, Huss attributes its appearance to cerebral congestion. In the Pneumonia of old people it is particularly common. Huss says that it is most frequent in patients who have been bled. It usually occurs during the height of the disease, and commonly makes its appearance at the time of the evening exacerbation of the pyrexia; it rarely continues during more than four or five days. I have known it to make its first appearance in the prostration following the subsidence of the fever.[2] In other cases I have observed it to commence immediately before the crisis, and to continue subsequently. Both of these events are, however, rare,—the period succeeding the crisis being more commonly characterised by a subsidence of pre-existing nervous symptoms.

Its appearance in a marked form is a sign of danger, and is indicative either of the prostration of the patient or of the severity of the disease. Grisolle says that three-fourths of his patients presenting this symptom died; but the treatment by bleeding to which they were subjected must be taken into account in estimating this degree of mortality, which certainly is not corroborated by my own experience, and though the symptom tends to occur in a large proportion of fatal cases, others may preserve a perfect intelligence to the last moments of life. The delirium, in fatal cases, tends to pass into an imperfect coma. A comatose condition independently of delirium is sometimes observed; it is most common in old people

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[1] Its frequency is variously estimated by different observers. Louis and Andral stated it at nearly 20 per cent.; but others—Grisolle, Briquet, and Huss—have shown that it does not appear in more than from 8 to 12 per cent. It often, however, occurs to a slight degree at night, and hence may fail to be noticed. Grisolle says that it is more common in males than in females, in the proportion of 21 to 6.

[2] The observations of Heintze (Arch. der Heilk. 1868) appear to show that the occurrence of delirium in Pneumonia is not specially connected with excessive elevation of temperature. In the cases observed by him it was much more frequent in cases of Pneumonia of the upper lobe than in that of the lower, in the proportion of 40·17 per cent. of the former to 25·5 of the latter. As regards season, it was more common in the cooler than in the hotter months of the year.

and in children,[1] and in the former there may be a complete pros-
tration of the mental faculties, extending even to a failure in the
pronunciation of words.[2]

In drunkards Pneumonia is so constantly associated with ner-
vous disturbance as to have led Huss to describe a special form,
the *Pneumonia Potatorum.* The delirium may assume the form of
active delirium tremens, with sleeplessness, delusions, and noisy
talkativeness, associated with tremors of the limbs and uncertainty of
pronunciation—symptoms which may sometimes appear with the
first invasion of the disease ; or in weakened patients, the subjects of
chronic alcoholism, the state may be one of profound prostration and
stupor, alternating with a low muttering delirium. In both these forms
the general signs of Pneumonia may be indistinct or may be masked
by the nervous symptoms, though in the first class the invasion
may be sudden and acute, and attended with rigors. Pyrexia is, how-
ever, present in both varieties, and is a valuable clue to the mischief
in the lungs.

Tremors are not uncommon in weakened patients independently of
delirium.

Convulsions are rare and quite exceptional in the adult. They are,
however, very common in children,[3] particularly under five years of
age, in whom they often attend the invasion of the disease, and they
are specially prone to occur if dentition is advancing or difficult. In
other cases they occur towards the fatal termination. They are some-
times general and epileptiform ; sometimes they appear only in the
form of spasm or rigidity of one limb, or of some of the muscles of
the face or the eyeballs ; occasionally also a stiffening of the muscles
of the neck, passing into opisthotonos and a tetanic state, has been
observed.[4] I have known a state of partial paralysis to remain
subsequently in the limbs affected.[5] When the convulsions are
general, and occur in the earlier stages of the disease, they are seldom
repeated ; but if this is the case, they generally end in a fatal coma.
Partial convulsive movements may, however, recur more frequently.

In other cases in children the cerebral disturbance may resemble
those seen in the earlier stages of tubercular meningitis, being marked
by prostration, headache, delirium, and strabismus—symptoms whose
deceptive character is further increased by attendant constipa-
tion. Barthez and Rilliet state that these symptoms are, however,
rarely accompanied by the automatic cries, by the sighing respira-
tion, the grinding of the teeth, or by the expression of indifference,

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[1] Grisolle relates a case of a young adult who remained perfectly insensible without
movement for twenty-six hours, but finally recovered.

[2] Hourmann and Dechambre, loc. cit.

[3] Barthez and Rilliet give to the affection of the nervous system in children the title
of "Pneumonie Cérébrale," which they subdivide into "Pneumonie Eclamptique" and
"Pneumonie Meningée."

[4] Weber, Path. Anat. des Neugeborenen und Sänglinge, ii. 61. These symptoms were
attended by inflammatory changes in the cerebro-spinal arachnoid sac.

[5] This, according to Barthez and Rilliet, is very rare.

and by the rapid changes of colour which characterise tubercular meningitis. Ziemssen, however, remarks that all these may be exceptionally observed, and that the coma may be so deep as almost to simulate death.[1] The *collective* appearance of this group of symptoms is fortunately of extreme rarity in the Pneumonia of children.

Disturbances of vision occurring suddenly, with undue sensitiveness to light, a false colouring of surrounding objects, and associated with a dilated condition of the pupils, have been occasionally observed. In these cases ophthalmoscopic examination has revealed undue distension of the veins of the retina; these symptoms disappeared soon after the resolution of the Pneumonia.[2] Deafness was observed in one case by the late Dr. Hillier,[3] and this symptom may at times add to the difficulty of diagnosis from typhoid fever. It does not, however, appear to be a common complication.[4]

The urine is diminished in quantity and increased in specific gravity during the acute period of the disease. The decrease in water may reduce the amount passed to little more than one-half the normal quantity. At the same time the excretion of urea is vastly augmented, amounting sometimes to 85·5 grammes or 1,326 grains in twenty-four hours,[5] though usually the amount varies between 35 and 55 grammes (761 and 858 grains).[6] This large amount of excretion necessarily represents destruction of tissue, for it is found at a period when very little food is taken. It usually reaches its height during the pyrexial period, increasing daily in amount until shortly before the crisis, though differences are observed in the period at which the maximum is attained. After the crisis, in spite of an increase of food, the amount may fall within one or two days, to or below the normal standard. In other cases an excess may be passed for some days during the period of resolution,[7] and the normal amount may only be attained on the fourteenth day.

The uric acid is also increased, and probably to a greater proportionate degree than the urea, and generally during the pyrexial period. It may reach at the crisis the amount of 37·7 grains,[8] or even the enormous amount of 103 grains[9] excreted in twenty-four hours. Like

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[1] In a case where this was observed by Ziemssen, the coma ceased with the crisis on the fifth day.

[2] Sichel, Gaz. des Hôpitaux, June 1861. Seidel, Deutsche Klinik, 1862, p. 269.

[3] Dis. of Children, pp. 40—42.

[4] Griesinger (Bleuler, loc. cit.) met with it five times in 228 cases.

[5] Parkes on Urine, 271. There will be found here a complete list of authors who have investigated this subject.

[6] The estimates of the normal amount of urea vary considerably. The normal daily average for an adult man under ordinary conditions of life may probably be regarded as 500 grains.

[7] Dr. Parkes (loc. cit.) says that he has found 50 or 60 grammes per diem during the period of resolution.

[8] On the tenth day. Zimmermann, Prager Viertel jahresch. 1852, vol. xxxvi. p. 118. The average normal amount appears to be from 6 to 9 grains daily.

[9] Huss, loc. cit. p. 47. This amount must be regarded as very exceptional, as would appear from other analyses given by the same author.

the urea, an excess may continue to be passed for some days after the pyrexia has disappeared.[1]

Large deposits of urates tend to occur during the whole period of the disease.

The sulphuric acid also appears to be slightly increased; the phosphoric acid is lessened, and the free acidity is said to be diminished.[2]

The chloride of sodium is markedly diminished, and sometimes its excretion is entirely suppressed during the height of the disease, even when hydrochloric acid or chloride of sodium is taken internally.[3] The hydrochlorate of ammonia continues in some cases to be excreted. The chloride of sodium reappears during resolution, and may for some days after be passed in excessive amounts, showing that it has been retained in the system; and the excess of chlorides may persist in the urine after that of the urea has ceased.[4]

Rigler has found that iodide of potassium, when given internally, is also retained in the system during the height of the disease, but that during resolution it is excreted by the urine.[5]

In some very exceptional cases the urea and uric acid appear to be retained in the system during the febrile period, even when there is no albuminuria, and are excreted in large quantities during convalescence, forming a sort of pseudo-critical discharge.[6] Dr. Parkes states that these patients are more liable to diarrhœa during convalescence, and that possibly some elimination of the retained matters may then take place by means of the intestinal mucous membrane. Patients presenting these phenomena of retention are also liable to a more protracted convalescence than those whose urinary excretion is large throughout the disease.

During convalescence the amount of water passed is increased, but that of the urea tends to fall below the normal amount, while the chlorides, as before stated, are commonly increased in quantity.

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[1] Dr. Parkes considers that this may probably be due to some of the uric acid being retained in the system, owing to its being less easily got rid of than "the diffusible urea." Zimmerman (loc. cit.) for the case quoted above gives the following averages :—

Stadium Incrementi	15 grains per diem.
Crisis	37·7 ,, ,,
Stadium Decrementi (21 days—average)	13·5 ,, ,,
First 7 days of Stadium Decrementi .	21·26 ,, ,,
Second 7 days ,,	11·9 ,, ,,
Third 7 days ,,	8·29 ,, ,,

[2] For these statements the author is indebted to Dr. Parkes's work. Huss, however, says that both these acids are diminished, at least in the form of their salts.
[3] For the chief investigations on this point see Redtenbacher, Zeitsch. der K. K. Gesellsch. der Aerzte zu Wien, 1850, by whom the discovery of this peculiarity was first announced; and Dr. Lionel Beale (Med.-Chir. Trans. xxxv.), by whom this subject was further investigated; also Dr. Parkes, loc. cit.
[4] For remarks on the relative excretion of the chlorides in the urine and sputa, see Appendix B.
[5] Beiträge zur Statistik der Pneumonie ; Wien Med. Woch. 1858, No. 48 (Canstatt's Jahresb. 1858).
[6] Parkes, loc. cit.

Albuminuria, usually slight in amount, is a more frequent complication of Pneumonia than of almost any acute disease, except typhus[1] It is found commonly during the height of the disease, more rarely during convalescence, but it may appear for the first time as late as the twenty-third day. In most cases it must be regarded as one of the general phenomena of the disease, due probably to the kidneys being affected by the same cause which sets up the inflammation in the lungs. Its presence is also indicative to a certain degree of the intensity of the cause, for cases in which it occurs are generally more severe in their character and more fatal in their issue than those in which it is not found.[2] It is very common also in the Pneumonia which appears as part of the general phenomena of some morbid blood poison, as in diphtheria and other conditions, to which further allusion will be made (see Pathology). It is very commonly attended with epithelial casts and sometimes with blood in the urine.

Bile pigment is not infrequent. The biliary acids are less common. Fibrine and cystine have also been found. The vesical mucus is increased, and the urine tends to decompose early (Dr. Parkes). I have observed retention of urine in one case associated with severe cerebral symptoms.

The skin is pungently hot, but many variations are observed with respect to perspiration. It may appear shortly after the rigor and subsequently give place to a dry pungent heat, or the skin may be dry until the crisis is attained, or perspirations may continue throughout the entire course of the pyrexial period. Andral thought that sweating was a favourable sign, but I have observed it more than once in fatal cases, and even in those where the temperature has not been markedly elevated. Profuse sweating usually attends and follows the crisis.

Louis remarked that sudamina were rare in Pneumonia.[3] They have been abundant in three of the cases which I have observed; a few also may often be seen when sweating is copious.

Herpes is a very common complication.[4] It most usually appears on the face, and particularly about the lips and angles of the

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[1] Parkes, loc. cit. Dr. Parkes quotes the following statistics. He found it in 6 of 13 cases, or in 46·1 per cent. ; Finger in 15 of 33, or in 45·4 per cent.; Becquerel in 9 of 21, or in 42·8 per cent.,—collectively representing 30 cases of albuminuria out of 67 cases of Pneumonia, or a ratio of nearly 45 per cent. Metzger, however, did not find it once in 48 cases. In 32 cases which I have analysed, it was found ten times, or in rather more than 31 per cent. ; Martin Solon and Ziemssen each found albumen only twice in 24 cases.

[2] In seven non-albuminous cases, Dr. Parkes met with only one death; while in five where albumen was present during the height of the disease, three died. Of the ten cases in which I find albuminuria to have been present, five died, but in one of these the disease of the kidneys was probably of old standing. Griesinger (Bleuler) found albumen in the urine in 63 out of 121 cases. Of these, 42 recovered and 21 died. In 22 cases where the amount of albumen was considerable, 8 died.

[3] Fièvre Typh. ii. 111.

[4] Ziemssen observed it in half of the cases of children under his care. Geisler ("Ueber die prognostiche Bedeutung des Herpes bei der Pneumonie," Arch. der Heilk. 1861, ii.) found it in 43·2 per cent. of 421 cases in Wunderlich's wards. In cases under my own care it has been less frequent than this.

mouth, but it may occur occasionally in other situations.[1] I have seen a tonsillitis having the characteristic appearances of the herpetic form appear on the fifth day of a pneumonia. It seldom appears before the third or fourth day, but I have known an eruption which from the description I concluded to be herpes precede the pneumonia by a period of some weeks, the patient remaining out of health in the interval. It may also appear during the crisis, and, in rare instances, during convalescence.[2]

The face, as has been stated, is flushed, particularly over the malar bones.[3] The flush may be bright in tint, or it may tend to a cyanotic or violet tinge, particularly in children. With the flush there is, however, usually an opacity or earthy tint of the skin around the eyes and lips. In rare cases the whole surface of the body may be of a bright-red tint, so as even to give rise to the suspicion of the presence of one of the eruptive fevers.[4] The flush tends to disappear with the progress of the disease; occasionally, and particularly in children, and sometimes in old people, there may be an earthy pallor throughout, which may be attended with a bluish tinge of the eyelids. Pallor of the face is most commonly observed during the crisis.

The *temperature*[5] of the body in Pneumonia has only been made the subject of accurate thermometric observations within the past twenty years, though many of the more important facts bearing on this subject had been previously stated by earlier observers.[6] It is, however, to Von Baerensprung,[7] Traube,[8] Zimmermann,[9] Wunderlich,[10] Thomas,[11] and Ziemssen [12] that we owe the revival of observation and most of our accurate knowledge on this subject.[13]

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[1] Thomas has observed it around the anus. (Arch. der Heilk. viii. 478.)

[2] Six days after the resolution of the fever. (Thomas, ib.)

[3] Unilateral flushing of the cheek on the affected side, and attended with a higher temperature than on the opposite side, was described by Gubler (Union Méd. 1857) as very common in Pneumonia and also in other pulmonary affections, and was attributed by him to the implication of the pulmonary branches of the sympathetic plexus. Other observers, as Barthez and Rilliet, have controverted this opinion, and have shown that the cheek on the side opposite to the affected lung may show an excess of hyperæmia. Jaccoud (loc. cit. p. 28) observed in an attack of Pneumonia in his own person, that a local flush, attended by a disagreeable sensation of heat in the cheek on the side opposite to the affected lung, preceded the Pneumonia for twenty-four hours, during which time, with this exception, he felt in perfect health. The Pneumonia then commenced suddenly with rigors. He states that he has met with five similar instances.

[4] Barthez and Rilliet (loc. cit. i. 522).

[5] In all ensuing statements on this subject, the temperatures quoted will be those of Fahrenheit's scale. Quotations from other observers have been reduced to this standard.

[6] Thus Donné (Arch. Gén. de Méd. 1837) observed a temperature of 103°, and Roger, in a more extended series of researches (Arch. Gén. de Méd., Sér. iv. vol. vi.), stated that Pneumonia had a higher temperature than almost any other disease, and that in the majority of cases this exceeded 104° Fahr.

[7] Müller's Archiv, 1851-2.

[8] Annalen der Charité, i. ; Ueber krisen und kritischen Tagen.

[9] Various writings in "Med. Zeit. des Vereins für Heilkunde im Preussen," specially in "Prager Vierteljahresch.," 1852.

[10] Various papers in "Archiv für physiol. Heilkunde;" "Das Verhaltniss der Eigenwärme im Krankheiten."

[11] Archiv für Heilkunde, 1864-5.   [12] Pleuritis und Pneumonie im Kindesalter.

[13] Among English authors the most valuable observations are those by Dr. Parkes,

One of the most marked features of Pneumonia,[1] which is almost sufficient to distinguish it from other diseases, is the sudden and considerable rise of temperature which marks its invasion, and which, with some exceptions, is then maintained, with slight morning remissions and evening exacerbations, throughout its course until a crisis occurs. The rise of temperature during the rigor is common to most diseases in which this phenomenon occurs,[2] but its subsequent maintenance at a very high standard during the succeeding first hours and days of the disease is limited to a small class of inflammatory affections.

An instance of this has been already given. I have known a case in which the temperature had reached 105° within a few hours of the first feeling of illness, although the usual rigor was absent; and others may be quoted from different observers who have had an opportunity of witnessing the earliest phenomena of invasion. Thus Zimmermann[3] relates a case where, after prodromata of a week's duration, the temperature within three hours after the initial rigor reached 102°, and within twelve hours it attained the height of 104°. Thomas[4] observed a temperature of 105° within nine hours of the invasion; Ziemssen, within four hours after the initial vomiting in a child, found a temperature of 102·5°; within twelve hours this had reached 104·6°, and within twenty-four hours the temperature was maintained at 103·5°.

The highest temperatures are most commonly observed on the second or third day of the disease, but exceptions to this rule are not infrequent. In some instances the maximum temperature, preceded by a very sudden rise of from one to two degrees Fahr. above the previous average, may occur immediately before the crisis. The highest recorded temperatures in cases ending favourably are 106·7° in the rectum[5] (Ziemssen), and 107° (Kocher), but they rarely exceed 105° or 106°. In fatal cases, however, there may be a considerable rise before death, as to 106·9°, 108·9° (seventh day), or

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Med. Times, 1866; by the late Dr. Waters, St. Barth. Hosp. Rep. vol. ii.; Dr. Compton, Dublin Quarterly Journal, xlii.; Dr. Grimshaw, ib. 1866; and Dr. Maclagan, Edinb. Med. Journal, 1869.

[1] Grisolle (loc. cit. 163) says that in some cases the course of Pneumonia is apyrexial throughout, though the physical signs and rusty sputa are present. Grisolle's statement is made apparently irrespectively of thermometric observations. Wunderlich, however, repeats the statement (Eigenwärme im Krankheiten, p. 337). Such cases must, however, be excessively rare, and require data as to the day of the disease upon which they came under observation. The majority of cases in hospital practice are rarely admitted before the third day, and it must be remembered that even at this early period the temperature may in some cases fall from a pyrexial height to the normal standard. I have never seen a case of Pneumonia unattended by pyrexia free from this suspicion.

[2] This was observed by De Haen, in Intermittent Fevers, Rat. Medendi, Ed. 1761, i. 117.

[3] Prager Vierteljahresch. 1852, xxxvi. p. 97.

[4] Archiv für Heilk. 1864.

[5] This was observed on the sixth day in a child who at the time was sweating profusely.

even to 109·4° (fourteenth day—Thomas[1]); a slight post-mortem rise is also occasionally observed. In the fatal cases which have come under my own observation this ante-mortem rise has not been noticed, but in most of these the temperature had been only moderate throughout. The higher temperatures, according to my own experience, are, however, rather the exceptions than the rule.[2] In the majority of cases it has seldom exceeded 104°, and a large number run their course without the temperature of 103° being attained. As a general rule the milder cases are those in which the pyrexia is least, but cases may end fatally in which the temperature has barely exceeded 102°. In old people especially, in whom Pneumonia is comparatively the most fatal, the temperature is very commonly lower than in adults.

After the invasion the pyrexia generally runs a certain definite course, with a series of regular daily exacerbations and remission, which commonly occur respectively in the evenings and mornings, representing in this respect, though with some irregularity, an exaggeration of the normal diurnal variations,[3] and which according to their relative extent have given rise to various classifications.[4] Usually the daily course is that the morning temperature from 6 to 9 A.M. is the lowest, but it seldom falls more than 1° or 1·8° Fahr. below that observed in the evening, and the temperature at these periods of remission never, or only in the most exceptional cases, reaches the normal standard. In the forenoon or early in the afternoon the fever again increases, commonly reaching its maximum intensity early in the evening, or sometimes even at mid-day.[5] From this point the temperature falls towards midnight, when a second slight exacerbation occasionally occurs, which does not however reach the same height as that of the afternoon.[6] Subsequently to this the temperature continues to

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[1] This terminal elevation of temperature is sometimes preceded by a marked remission. It is sometimes gradual, extending over a period of from twelve to twenty-four hours, but it may take place very rapidly, *i.e.* within six hours. The temperature has been known to rise on the supervention of severe brain symptoms from 101·7° to 108·7°, or 7 degrees Fahr.

[2] Out of twenty-seven cases whose temperature has been carefully taken throughout, in one only was a temperature of 105·8° attained on the sixth day, the crisis occurring on the eighth day. Griesinger (Arch. der Heilkunde, i.), out of seventy-two cases, only observed the temperature higher than 103° Fahr. in nineteen.

[3] See Von Baerensprung, Müller's Archiv, 1851, pp. 160 et seq.; Ib. 1852, p. 251.

[4] Those proposed by Thomas and Wunderlich are as follows :—
    (*a*) A subcontinuous course, with daily variations of from ⅔° to ⅘° Fahr.
    (*b*) Subremittent, with daily variations of ⅘° to 1·5° Fahr.
    (*c*) Remittent, with variations from 1·5° to 2·5° Fahr.
    (*d*) Intermittent, a very rare form, with complete apyrexial periods in the daily course.
There is a form of Pneumonia described as accompanying intermittent fever where there also appear to be complete apyrexial periods corresponding to the type of the fever.

[5] This occasional irregularity renders a mid-day observation on the temperature necessary in all cases when scientific accuracy is required. In fact, unless frequent observations are made, the period of the *maximum* elevation of temperature may escape observation.

[6] This second exacerbation may sometimes be anticipated ; that is, when the afternoon exacerbation occurs early, a rapid fall may take place until early in the evening, and the second rise may take place early in the evening instead of at midnight.

sink during the night until an early hour the following morning, from which a gradual rise takes place, culminating in the exacerbation of the succeeding afternoon. In very rare cases the rise of temperature takes place early in the morning, when the highest temperature is observed, under which circumstances the corresponding remission is noticed at mid-day, or a continuous fall takes place until the evening.

During the course of the acute disease the morning remissions and evening exacerbations maintain in typical cases a very uniform standard of temperature until the period of the crisis is attained, unless fresh extensions of the pneumonic process occur, when a sudden increase of the temperature may be usually observed. In the earlier periods of the disease or during the *stadium incrementi* a more marked remission may occasionally occur, and may even be repeated more than once. This is followed in some cases by an intenser exacerbation, but in others the temperature of the succeeding rise falls below the average standard of the case. When an exacerbation of the fever follows this remission, it is also frequently attended by an extension of the pneumonic process or by a secondary inflammation of some other organ.[1]

The pyrexia tends to subside abruptly by crisis or gradually by lysis, the resolution by crisis being however the most usual form, and the rapidity with which this takes place in typical cases is again almost peculiar to this disease. The thermometric phenomena of the crisis may commence either at the period of the morning remission or of the post-meridial exacerbation. If at the former, the temperature, which on the preceding evening may have maintained its previous height, is found on the following morning to have fallen to the normal or nearly to the normal standard, and the succeeding exacerbation on the following evening is less by 1 or 2 degrees Fahr. than those previously observed. From this period a gradual fall of temperature ensues, and within forty-eight hours from the commencement of the crisis it has usually reached the limit of health, or it may have fallen below it, and after this no further elevation ensues.[2] The extent of the fall of temperature is sometimes very remarkable when the fever has been severe, amounting even to 9·7° Fahr. in sixty hours.[3] This is sometimes intensified by the fact that when the fever has been high and the patient is weak, and in children particularly, the temperature may sink during the critical defervescence to 1°, 2°, or 2·5° below the normal, and may continue at this low point for forty-eight or seventy-two hours. I have observed in a child a temperature of 96·5° (axilla) with a cold skin and profuse perspiration maintained in spite of artificial warmth for forty-eight hours. Such cases in children, however, do not

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[1] Kocher, Behandlung der croupösen Pneumonie mit Veratrum Preparaten ; Würzburg, 1866.

[2] Griesinger (Bleuler, loc. cit.) found this rapid fall of temperature in 112 out of 146 cases. The normal temperature was reached within twelve hours in 37 cases ; within twenty-four hours in 32 cases ; within thirty-six hours in 43 cases. In 41 cases the fall of temperature was more gradual.

[3] Ziemssen, loc. cit. 211.

commonly end unfavourably. Variations in the phenomena of the crisis are, however, not uncommon. Sometimes immediately before it occurs the temperature may rise to a higher point than those previously observed. In other cases, for twenty-four or forty-eight hours previously, both the morning and the evening temperature may show a lower range before the final rapid decline takes place. In some, again, the crisis is marked rather by successive falls of temperature during the periods of remission, those of exacerbation maintaining during twenty-four or forty-eight hours the same height as before, but finally participating in the decline—a course which may be regarded as presenting simply a modification of the ordinary rhythmical progress of the disease.

Sometimes, after the crisis has distinctly appeared, the fall of the temperature is suddenly checked, and a temporary exacerbation may occur, attended by a cessation of the critical perspiration and by a return of the restlessness and of the other febrile symptoms.

For some days after the disappearance of the fever there is also a tendency to temporary trivial exacerbations from slight causes, such as a meal or a slight exertion; but these, within certain limits, do not interrupt the progress of convalescence.[1]

The course of the fever is, however, subject to other variations, which are due commonly to the progressive invasions of other portions of the lung or of the opposite lung, and the crisis may be disturbed by a relapse.

The former may occur while the fever is still present. They are often marked by an increased intensity of the succeeding exacerbations, which may give the pyrexia a remittent type.

Relapses most commonly occur within the first three or four days succeeding to the crisis.[2] I have, however, known a relapse take place as late as the sixteenth day, when the temperature had been natural since the eighth day.[3] They are marked by a sudden rise of temperature from the normal or subnormal standard previously attained. The duration of the pyrexia in these relapses is, however, commonly shorter than that of the primary attack, usually terminating within three or four days; but cases are recorded where even a third relapse has ensued.[4] It is therefore very important to maintain ther-

[1] Monthus (loc. cit.) remarks that the apyrexial period following an attack of double Pneumonia is marked by a rather higher temperature, and more readily shows slight subsequent exacerbations than when the Pneumonia has been unilateral.

[2] Monthus, loc. cit. 206.

[3] The duration of the pyrexia in the second attack was only two days.

[4] See a case by Ziemssen, p. 186, of Pneumonia of the upper lobe. The maximum temperature of the original attack was 102° Fahr. On the ninth day the temperature fell to normal. On the tenth day there was a return of the fever with invasion of the middle lobe, and with a temperature of 104·9°. On the fourteenth day a second remission of the pyrexia took place, followed on the fifteenth by a return of the fever (temperature 102·7), and with invasion of the lower lobe. The final crisis and permanent recovery occurred on the eighteenth day. In another case by the same author, with Pneumonia of the left lower lobe, the crisis occurred on the fifth day. On the sixth there was a severe return of the fever, followed on the eighth day by the physical signs of consolidation of the right upper lobe, while the resolution of the lung first

mometric observations for some days after the normal temperature has
been attained—the more so as relapses with invasion of other portions
of the lung are seldom attended with a return of the initial rigors, and
the increased temperature may give the first indication of the extension
of the disease.

In other cases the temperature falls by a gradual lysis, which, in
some cases that I have observed, has only reached the normal standard
on the twelfth or fourteenth day.

In a third series, again, the crisis is incomplete, and the course of the
pyrexia is protracted. There is very often noticed on one of the days
intervening from the seventh to the tenth inclusive, a marked fall
both of the morning and evening temperature; but this does not
reach the normal, and on the succeeding days pyrexia persists, though
not usually at its previously high standard. A slow defervescence then
ensues which may be protracted over two or three weeks, and is
attended with a somewhat irregular course of the temperature—that
in the morning being often nearly normal, while in the evening
it may be on some days 100° and on others 102° Fahr., occa-
sionally rising to 103° or 104°, and on the succeeding evening it
may again only be 100°. These cases are generally attended with a
protracted disappearance of the physical signs—the consolidation and
bronchial breathing with fine râles lasting, together with the pyrexial
state, for three weeks or a month, but gradually disappearing and
ending in perfect recovery.[1]

The circumstances determining a more protracted course of the
pyrexia are not always clearly discoverable. Cases where bronchitis
passes into Pneumonia, and which belong rather to the clinical cate-
gory of broncho-pneumonias, often evince this tendency; but I do not
think that this peculiarity is sufficient under all circumstances to
remove a case in which it is observed from the category of the pri-
mary form, as it is occasionally seen when the mode of invasion and
the earlier course are typical of this condition; and it must therefore,
I think, be regarded as a somewhat exceptional variation.

The cases in which I have observed this course are most commonly

affected continued unimpeded. The final fall of temperature began on the eleventh
day and continued through the twelfth, when convalescence was re-established. Grisolle
says that relapses occurred with him in the proportion of once in 28 cases. Briquet
met with 16 instances in 92 cases. Grisolle quotes a case in which three consecutive
relapses took place, the last being on the twenty-seventh day. Commonly the course
of the relapse is shorter than that of the first attack, rarely lasting more than three
or four days. In the case, however, quoted, each attack lasted nine days, and the last
was very severe. (There is some confusion in Grisolle's statement with respect to the
duration of the relapses.)

[1] In one case under my own observation, a boy aged 15, previously in good health,
got chilled. Pneumonia supervened with rigor on the following morning; admission
on the third day of disease with well-developed Pneumonia of the lower two-thirds of the
right lung, and severe gastric catarrh. Temperature on fourth day, 105°; on the eighth
day it fell to 99°; on the ninth and tenth days it was 99° and 98·4°; on the eleventh
day it rose to 100° without any discoverable increase of the Pneumonia. It then
fluctuated between 100° and 102°, reaching to 103° on the fourteenth day, and only fell
gradually to the normal on the thirty-fifth day. The physical signs only completely
disappeared by the sixtieth day.

those which are accompanied by an extreme degree of gastric catarrh, or when the Pneumonia attacks persons of weakly constitutions, or those of previously dissipated habits. In some cases also where bleeding has been practised the recovery has been slow.[1] A very widespread opinion now exists that venesection tends to retard convalescence. The presence of tubercle or the tubercular diathesis appears also to protract the course of acute Pneumonia. Such patients may in many instances recover entirely from the inflammatory consolidation, but in others the resolution is imperfect, and the disease, although in rare instances, passes into the condition of a chronic tubercular Pneumonia.[2]

Pneumonia of the apex is said by Ziemssen and Bleuler to tend to maintain a high temperature during a longer period than that of the base[3] and that in non-tubercular patients, though the protracted course may give rise to the suspicion of this complication. This, however, is not invariably the case, for I have known the crisis to occur in a well-marked case of Pneumonia of the apex as early as the fourth day. Ziemssen thinks that such cases may be distinguished from tubercular Pneumonia by the constantly maintained high temperature; but my own experience would show that this sign cannot be relied on, since I have observed that the elevation of temperature in cases of protracted simple Pneumonia is not always continuous, while it may be so in some cases of tuberculosis and of tubercular Pneumonia.

The co-existence of pleuritic effusion certainly tends in some cases to render the thermometrical crisis incomplete and to protract the period of defervescence. It also, as might be expected, delays the disappearance of the physical signs; the complication with pericarditis has a similar influence in the crisis. Ziemssen remarks that neither pleurisy nor pericarditis, when occurring in the course of Pneumonia, have any necessary tendency to raise the temperature above the standard of the individual case.

The period of the crisis has been a subject of considerable discussion and of careful thermometric observation. The recognition of this

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[1] This was the case in that by Zimmermann before quoted. The case was peculiar in its course. The temperature on the first day was 104° in spite of VS to 2 lbs. and repeated on the second day to 14 oz. Up to the third day there were only the physical signs of congestion, but the respiration was slightly bronchial in one place. On the third day there was a distinct remission in the morning (99·8²), but followed by an evening exacerbation to 105·8°. On the fourth day, rusty sputa, dulness, and bronchial breathing appeared in the lung. A second imperfect crisis occurred on the ninth day, with a subsequent elevation of temperature on the tenth, reaching 103° on the seventeenth day, and with irregular intermissions maintaining a temperature of 100° to the twenty-fifth day.

[2] This course is, however, very rare in the acute primary disease. Most of the forms of tubercular Pneumonia run the course of catarrhal or broncho-Pneumonia.

[3] See a case by Ziemssen (loc. cit. pp. 180-2) of Pneumonia of upper lobe, in a child aged nine months. The pyrexia lasted thirty-one days, and the physical signs only disappeared three weeks after the subsidence of the fever. Bleuler (loc. cit. p. 19) states that of the cases observed by him and Griesinger when the inflammation affected the apex of the right lung, in one only did the fall of temperature occur before the sixth day, and in three-fourths of these cases it took place after this date, while in more than half the cases of Pneumonia of the base defervescence ensued from the third to the fifth day

tendency has been common to many observers, and it was pointed out by Laennec. Andral[1] thought that the seventh, fourteenth, or twenty-first days were the most common periods of its occurrence—supporting the doctrine of special critical days advanced by Hippocrates. Grisolle disputed this opinion. Traube (Ueber krisen und kritischen Tagen) has again revived it, and has asserted that in acute diseases, and especially in Pneumonia, the crisis usually occurs on the third, fifth, seventh, ninth, or eleventh days, and that therefore it has a preponderating tendency to appear on uneven days. This, however, has been denied by different observers, whose observations show that the crisis is by no means so constant on the uneven days as Traube believed, but that in a large proportion of cases, amounting respectively to 20 and 25 per cent. and collectively to 46 per cent. of the whole number, it tends to occur on the fifth and seventh days.[2]

The cases of which I possess sufficiently accurate thermometric observations give very similar results, though in smaller numbers. Out of twenty-seven cases ending favourably, a distinct thermometric crisis occurred in eighteen, and two more were admitted on the fifth and eighth days respectively with the physical signs of Pneumonia, but with a normal temperature, which was maintained subsequently. These, therefore, may justly, I think, be added to the above, making the proportion of cases terminating by crisis, as compared with those not thus ending, as twenty to twenty-seven.

The following were the days[3] in which a crisis was observed :—On the fourth day, one case ; on the sixth, one ; on the seventh, six ; on the eighth, two ; on the ninth, four ; on the tenth, two ; and on the eleventh day, two cases. The period of complete defervescence varied from twelve hours (four cases) to seventy-two hours (one case). In the remainder it varied from twenty-four to forty-eight hours.

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[1] Clin. Méd. iii. 516.
[2] The days of crisis observed by Wunderlich (Spec. Path. Therap. Abth. iii. B. ii p. 334), Ziemssen (loc. cit. 174), Thomas (Arch. der Heilk.), and Bleuler (loc. cit.) may be best expressed in a tabular form :—

CRISIS, NUMBER OF CASES.

Day of Disease.	Wunderlich.	Ziemssen.	Thomas.	Bleuler.	Total.
1st	0	0	0	0	0
2nd	0	0	2	0	2
3rd	10	9	6	6	31
4th	11	3	6	13	33
5th	14	31	11	22	78
6th	14	5	5	26	50
7th	19	35	10	32	96
8th	4	4	4	24	36
9th	3	9	0	12	24
10th	0	0	2	6	8
11th	0	8	0	1	9
12th	0	0	0	3	3
13th	0	3	0	1	4
14th	0	0	0	0	0
	75	107	46	146	374

[3] I have reckoned the day of invasion as the first, the next day as the second day.

Two other cases terminated by gradual lysis, one on the twelfth and another on the fourteenth day, the temperature gradually falling to the normal.

In five others the duration was protracted without complications, which would account for the persistence of the pyrexia, except in one instance, where there was considerable pleuritic effusion. In this case an imperfect crisis took place on the tenth day, but the temperature remained elevated until the forty-sixth day. Of the remainder, three recovered perfectly, though the pyrexia only ceased on the twenty-fourth, thirtieth,[1] and thirty-fifth days respectively.[2] In the fourth there was, however, a suspicion of tuberculosis. The Pneumonia, which had invaded the whole right lung, and which was complicated with pleurisy, resolved imperfectly, and occasional pyrexia remained until the eighty-first day.

As far as I have observed, I do not think that cases where the temperature is much elevated, *i.e.* above 104°, necessarily have a longer duration than those in which the pyrexia is less marked. The pyrexia in the former may end rapidly by an early crisis, and in the latter its disappearance may sometimes be considerably protracted. My own observations would also tend to confirm Thomas's opinion, that the extent of lung affected does not necessarily delay the appearance of the defervescence, though cases supporting the contrary opinion, which has been advanced by Ziemssen, may sometimes be met with.

Together with the disappearance of the fever, the aspect of the patient markedly changes. The flush disappears, and profuse sweating is almost constantly observed.[3] The face may be pallid and sunken, and, as before stated, the general condition may be one of such intense collapse as to lead to immediate fears of a fatal issue, which indeed sometimes occurs at this period.[4] The pulse becomes small and often dichrotous, and generally falls in frequency. It seldom, however, attains the normal standard, and is liable to irregular exacerbations for some days later, quite irrespective of any corresponding variations of temperature. Children particularly may be for hours partially unconscious and almost incapable of being roused, with a cold skin bathed in colliquative perspiration.[5] A catarrhal flow from the nose is sometimes also observed in children at this period simultaneously with the perspiration.

The respiration at the same time falls in frequency. The pain in the side, if this has persisted up to the period of the crisis, disappears or

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[1] This case was a man of dissolute habits. An imperfect crisis took place on the ninth day. The general symptoms were very severe, with profuse puriform sputa after the second week, giving rise to strong suspicions of grey hepatization.

[2] This case has been already alluded to (see note, p. 648). There was in this case an imperfect crisis.

[3] Herpes also may appear as a critical phenomenon at this time.

[4] I have seen two cases of this nature.

[5] An excellent and life-like description of this condition is given by Ziemssen, loc. cit. 167.

is much relieved. The cough becomes looser; the expectoration loses its tenacity, and the rusty character diminishes, though it may not finally disappear until some days later. In its further course and during the resolution of the Pneumonia the sputa gradually assume a bronchitic character. The most marked appearance is, however, that of black pigment, which takes the place of the rusty tinge of blood, and the early appearance of which is a favourable sign. The amount of this pigment in some cases, when the resolution is retarded, is sometimes very considerable: I have seen the sputa during many days almost black from its presence.

Other phenomena are occasionally observed, some of which have been regarded as truly *critical*, that is to say, as in part conducing to the fall of temperature; others, however, must be looked upon as accidental, or as a result of the subsidence of the fever. Among the former, whose influence in really producing a fall of temperature must be considered doubtful, are hæmorrhage and diarrhœa. Hæmorrhage is occasionally observed in the form of epistaxis, more rarely as hæmaturia, and occasionally it proceeds from the bowels.[1] Diarrhœa is more common,[2] but it must be remembered that the crisis may take place without any of these events, and their appearance is as a whole decidedly exceptional, the only constant critical discharge (with the exception of the changes in the amount of the urinary secretions) being that from the skin.

Erysipelas is mentioned as an occasional critical phenomenon.[3]

When the nervous system has been profoundly implicated during the pyrexial period, the symptoms of such disturbance also commonly disappear during the crisis. Delirium or extreme restlessness usually pass, particularly in children, into quiet sleep. In adult persons, and especially in those of dissipated habits, this may not be the case; I have seen symptoms closely resembling delirium tremens persist during forty-eight hours after the normal temperature has been reached and maintained.

The physical signs of the disease may begin to improve coincidently with the disappearance of the fever.[4] The commencement of the resolution, however, is more commonly observed after the first twenty-four or forty-eight hours of the apyrexial period. In some cases it is

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[1] I have only seen one case of this nature.

[2] Huss, p. 53, says that diarrhœa most commonly occurs on the seventh day in cases of Pneumonia characterised by severe gastric disturbances, but that the convalescence of such cases is usually protracted.

[3] Grisolle quotes from Serres a case of a patient who had several attacks of Pneumonia, each terminating in an attack of erysipelas. I have only seen one such case. The erysipelas appeared three days after complete defervescence, and the resolution of the Pneumonia and the subsequent recovery of the patient were greatly protracted.

[4] Grisolle states that the improvement in the physical signs may precede the disappearance of the pyrexia. Grisolle's statement appears, however, to be made independently of thermometric observations. I have never seen a case where this occurred before a marked fall of temperature, and it must be remembered that Grisolle regards a rapid pulse as one of the phenomena of the fever. It has been already stated that the pulse may remain rapid after the fever has subsided.

so rapid that all the physical signs of the disease may have totally disappeared in twenty-four hours from the first appearance of improvement.[1] I have seen this in one case where the whole lower lobe has been implicated, and it may occur without any marked increase of the expectoration, or even when this has been scanty and quite insignificant in quantity. Indeed it may be said that, generally speaking, the proportion of exudation removed by expectoration must be small in comparison with the whole amount present in the lungs. Commonly, however, the course of resolution is more protracted. Grisolle states that all the physical signs had only completely disappeared in 37 out of 103 cases who left the hospital between the twentieth and the fifty-fifth days. Dr. Stokes gives the following results of 24 cases, dating from the commencement of resolution. In nine the physical signs had disappeared at the end of a week; in nine more at the end of fourteen days; in five at the end of three weeks; and in one they lasted a month. In 26 cases of which I have notes of the total [2] disappearance of the physical signs, their duration after defervescence was as follows:— In one case, two days; in three, three days; in one, four days; in one, five days; in one, six days; in three, seven days; in one, nine days; in nine, from ten to fifteen days; in five, from twenty to twenty-five days; in one, from twenty to thirty. Two other patients left the hospital with physical signs still remaining on the twenty-fourth and eightieth days.[3] In some of the cases of longer duration the Pneumonia was complicated with pleurisy, and when much effusion has been present some dulness at the base may remain almost indefinitely, as I have seen in one or two cases not included in this list. The co-existence of tubercles may also indefinitely protract the resolution. Patients whose health has been previously bad are also liable to a retarded resolution; but this is not always observed. The same tendency has been noticed in cases where the defervescence is not marked by a crisis, or only by an imperfect one.[4]

Dr. Stokes has observed that retraction of the chest walls may follow an attack of Pneumonia. This has been disputed by Grisolle and Woillez; but Dr. Walshe has seen it take place when the Pneumonia had been unattended by liquid effusion into the pleura.

[1] Ziemssen gives a case of a child where the physical signs had disappeared before the end of the eighth day of the disease.

[2] This includes the final disappearance of all râles as well as dulness and bronchial breathing. Crepitation or fine moist râles may, as has been before stated, often persist for days, or even weeks, after all other signs have disappeared. Probably the weakened resistance of the vascular coats leaves, during a lengthened period, a tendency to a certain degree of œdema, particularly in the lower portions of the lung.

[3] Bleuler (loc. cit.) gives the following periods of resolution in 150 cases:—One day, 5 cases; two days, 2 cases; three days, 4 cases; four days, 21 cases; five days, 21 cases; six days, 30 cases; seven days, 13 cases; eight days, 11 cases; nine days, 5 cases; ten to fifteen days, 18 cases; fifteen to twenty days, 6 cases; more than twenty days, 7 cases, among which were included 3 cases of Pneumonia of the right upper lobe.

[4] Ziemssen remarks that in cases where the crisis is early, resolution may be short, but my own experience has not confirmed this.

I have also observed it in one of the cases of protracted Pneumonia before mentioned.[1]

The recovery of strength and of flesh is generally very rapid. The appetite often returns almost with the cessation of the pyrexia. Wachsmuth observed in a patient whose loss of weight in four days amounted to a daily average of 24 oz. in the twenty-four hours, and in whom the loss of weight continued for forty-eight hours after the crisis, that in the succeeding four days nearly 2 lbs. were regained.[2] I have repeatedly observed that from 7 to 14 lbs. may be gained in weight during the first few weeks of convalescence.

An attack of acute Pneumonia is seldom succeeded by secondary diseases, except in patients liable to tubercle. Ziemssen has observed in children that œdema of the lower extremities may be caused by a pure hydræmia, independently of albuminuria, which, however, is sometimes present to a slight degree. Dr. Walshe has observed the same phenomenon associated with coagulation in the veins.

Gubler[3] and Macario[4] have each observed instances of general paralysis following Pneumonia, but these cases are fortunately rare.

The *termination* of Pneumonia is not, however, always favourable. It may end fatally, or it may give rise to local abscess or to gangrene of the lung, or finally it may pass into a chronic state.

In some cases, which may prove fatal during the acute stage, the pyrexia may persist to the last, and may, as before stated, increase rapidly towards the close of life. In others, however, no elevation of temperature occurs, and it may even sink to normal before the fatal termination: I have seen in one case, in a patient aged 62, death occur after the crisis had taken place forty-eight hours previously, and in whom during the first portion of this period the symptoms might on the whole have been considered favourable.

Most commonly death ensues during the acute period of the disease, when it is usually preceded either by intense prostration or by extreme dyspnœa. The pulse becomes small and extremely rapid and dichrotous, and the respiration is commonly greatly accelerated. Expectoration becomes difficult, or ceases, while large coarse metallic râles are heard in the trachea and larger bronchi, and fine and medium-sized râles, indicative of œdema of the lung, extend over the non-consolidated portions. The face becomes livid, the extremities cold, and the skin is often bathed in profuse perspiration, which is

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[1] In a boy in whom the pyrexia and physical signs lasted together sixty days (see note, p. 648), there was observed when he left the hospital some flattening inferiorly of the right (the affected) side. One month later, when he presented himself for examination, the measurements were : At nipple—right side, 12 inches ; left, 12 inches. At sixth rib—right side, 11 inches ; left, 11½ inches. There was also some procidentia of the shoulder on the right side. The amount of effusion present here was throughout extremely small, but some dulness still remained at the right base, attended with weak breathing.

[2] Zur Lehre von Fieber, Arch. der Heilk. 1865, p. 236. In this case the temperature had been very high, 106·1°, and the defervescence was gradual after the crisis.

[3] Arch. Gén. 1860-1.

[4] Gaz. Méd., Par. 1858. Huxham says : "I have seen in some cases (though few indeed) a complete paraplegia." (On Fevers, p. 183.)

colliquative when the temperature is low. A semi-comatose state supervenes towards the last, but in some instances intelligence is preserved to within a few minutes of the fatal issue. In children, coma or convulsions are very common. In old people death may often take place suddenly and unexpectedly.[1]

Sometimes, particularly in children, as described by Ziemssen, death may occur at a later stage. The fever does not maintain the high standard of the earlier periods of the disease, but persists together with the physical signs. The pulse remains accelerated, the skin becomes intensely pallid; emaciation, reducing the patient to the extremest degrees of marasmus, progresses rapidly; and the patient dies in the third or fourth week. In other cases there is observed an incomplete remission, followed by a return of the fever, and the patient gradually sinks in the course of the second week.

Some cases, however, presenting these characters lapse into a more chronic stage : the fever and physical signs may persist during many weeks, but the former may subside, while the lung remains permanently consolidated with signs of dilatation of the bronchi.[2]

No special condition of the lung is necessarily associated with a fatal termination in the earlier periods of the disease, but the red or grey hepatization, or even diffuse suppuration, may be found in different cases under circumstances which are otherwise apparently similar.

The termination in *abscess* is very rare. Huss says it only occurs once in fifty or sixty cases, and usually only in patients of bad constitution. According to this author, it is most commonly met with in males over forty years of age, and he states that it was more common when bleeding formed part of the treatment than it has proved since this was abandoned by him. The period of this termination, as determined by profuse purulent expectoration, has varied, according to Grisolle, between the fifteenth and twenty-eighth days. Profuse expectoration may continue for three months subsequently. The site of the abscess is usually at the apex; one case, however, has been recorded by Dr. Stokes, where a cavity in the midst of pneumonic tissue was found at the base of the lung. The signs of this condition have been already described.[3] Cases in which it occurs usually run a protracted course, though death ordinarily, according to Grisolle, takes place before the thirteenth week. Pyrexia persists, and the expectoration, which is at times intermittent, consists of large quantities of puriform matter. The pyrexia tends to assume the character of hectic fever, but from the rarity of the disease thermometric observations are wanting. Emaciation progresses as long as the fever remains, and many cases end fatally, sometimes with the signs of pyohæmia, in other instances by rupture of the abscess into the pleural cavity, and occasionally by sudden suffocation resulting from the filling of the bronchi with pus. Others, however, progress

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[1] Cruveilhier, Path. Anat., Liv. xxix.
[2] These cases will be again considered under the head of Chronic Pneumonia.
[3] See *ante*, p. 634.

more or less completely to recovery; in these the abscess cavity may either cicatrize, or it may remain patent but comparatively quiescent, and revealed only by physical signs more or less distinctly indicating its existence.[1]

The termination in *gangrene* is almost equally rare with that in abscess, and Grisolle has even doubted whether it is a cause or a consequence of the latter. Some well-authenticated instances are, however, recorded, and it appears that an epidemic constitution may at times predispose to its occurrence.[2] It commonly appears late in the disease; but it has been seen as early as the fifth day (Huss). In fifty-three cases of which I possess observations, I have found two instances of gangrene,[3] and in both these it was irregularly diffused though scattered spots of pneumonic infiltration. Its site, according to Huss's observations, is most commonly in the lower lobe, and it has almost invariably occurred in exhausted constitutions. Gangrene is much more common in tubercular Pneumonia. Its physical signs have been already described. In addition to these its advent is usually marked by a sudden and intense prostration of strength, with a rapid weak pulse and sunken countenance. The characteristic sputa are, however, the only positive signs, when developed suddenly in the course of a primary Pneumonia. It appears to be almost invariably fatal.

COMPLICATIONS OF PNEUMONIA.—Some of these affecting the kidneys and nervous system have been already described. Others, however, deserve mention.[4]

Laryngitis, though not mentioned by Huss, is an occasional complication. Grisolle quotes Serres as having collected the histories of ten cases, and Dr. Walshe says that œdema of the glottis may be one of the causes of a fatal termination.

Bronchitis is a more frequent complication. Grisolle says that it has occurred in one-fourth of his cases, that it is seven times more common in males than in females, and that it is most frequent in the winter months. It affects both lungs, though it sometimes appears in excess on the affected side. Its intensity varies, however, greatly in individual cases. Its presence, when general, increases the dyspnœa and the lividity of the face. It also renders the sputa more abundant and the cough more frequent. It is seen from Huss's

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[1] Of 20 cases, Huss states that 12 died, 4 recovered completely, and 4 only partially. A case of cicatrization of a supposed pneumonic abscess has been recorded by Dr. Stokes.

[2] Hughes (Guy's Hosp. Rep. 2d Ser. vii. 1848) found 28 cases of gangrene in 200 post-mortem examinations of Pneumonia. At one time it was noted that several cases of gangrene appeared during the prevalence of an epidemic of influenza, and that as many as six cases occurred in one week.

[3] See also notes to Section on the Morbid Anatomy of Pneumonia, "Gangrene."

[4] Under this head I only propose to treat of such complications as may appear secondarily to or simultaneously with Pneumonia. The following table from Huss gives a relative estimate of the frequency of other complications, and of their influence on the mortality. This table appears to include cases of both catarrhal and acute primary Pneumonia; but while some chronic diseases are mentioned, the omission of others, as

tables that it tends (at least when severe) to increase the mortality of the primary disease.[1]

Pleurisy is also very common. There are, indeed, very few cases of Pneumonia reaching the surface of the lung in which the visceral pleura is not implicated. Effusion, according to Grisolle, occurs in about 15 per cent. The amount of fluid is commonly in inverse ratio to the extent of lung implicated. Its signs are naturally, almost invariably, found at the base, whatever the site of the Pneumonia. Its influence on the pyrexia and on the progress of resolution has been already considered. Unless very considerable in amount, or when occurring on the side opposite to the pneumonic lung, it does not very materially modify the mortality. Under the latter circumstances, however, it may dangerously lessen the respiratory surface. Pneumothorax has been mentioned as an occasional complication; but its existence is very doubtful, and is entirely unsubstantiated by post-mortem evidence. Probably the tympanitic note occasionally heard over the non-consolidated parts has given rise to error in this respect.

Pericarditis, though a less common event, is a very dangerous complication. Huss's statistics show that it proves fatal in more than half

cancer, is remarkable. Huss, however, does not treat of the secondary Pneumonias complicating other diseases.

	Recoveries.	Deaths.	Total.	Percentage of Deaths.
Pleuritis	92	12	104	11·53
Bronchitis Capillaris, acute	120	20	140	14·28
Bronchitis Chronica	36	6	42	14·28
Emphysema Pulmonum	20	6	26	23·07
Tuberculosis Pulmonum	24	12	36	33·3
Pericarditis	10	12	22	54·54
Endocarditis	1	3	4	75·
Phlebitis after bleeding	0	2	2	100
Valvular Disease of Heart	16	7	23	30·43
Meningitis Cerebralis	0	2	2	100
Erysipelas Faciei	11	1	12	8·33
Catarrhus Intestinalis	110	13	123	10·56
Enteritis et Entero-Colitis, acute	31	6	37	16·21
Colitis Chronica	0	2	2	100
Icterus	21	2	23	8·69
Bright's Disease	20	26	52	50
Acute Articular Rheumatism	20	2	22	8·69
Intermittent Fever	60	6	66	9·09
Chlorosis	20	5	25	20
Delirium Tremens	144	36	180	20
Chronic Alcoholism	12	4	16	25
TOTAL	774	185	959	

[1] The inclusion of cases of Broncho-pneumonia in Huss's statistics must, however, be remembered.

the number of cases affected. In some cases it appears to originate in the same cause as the Pneumonia, or it may be caused by a direct extension of the inflammatory affection—(it may, however, occur, and apparently with about equal frequency, in pneumonias of the right and left side)—or, finally, it may in some cases be due to secondary septic effects resulting from the absorption of inflammatory products in the lung.[1] Its influence on the pyrexia and on the phenomena of resolution have been already described.

The evidence of other cardiac lesions secondary to Pneumonia is but slight, but in some cases there appears to be a tendency to the formation of fibrinous concretions in the cavities of the heart.

Icterus.—A slight icteric tinge of the conjunctiva is by no means uncommon. Distinct jaundice is also an occasional complication.[2] It may in some cases be produced by congestion of the liver, arising from the impeded circulation in the lungs; in others it is probably due to coincident gastro-duodenal catarrh. It is more common in the summer than in the winter months. It appears to be more frequently associated with Pneumonia of the right than with that of the left lung; but it must be remembered that the former is much more liable to be affected. The theory of its production by direct extension of the inflammatory action from the lung to the liver is now generally considered untenable.[3] I have met with one case in which icterus preceded the attack of Pneumonia; it usually, however, follows the invasion of the disease. According to Grisolle, the liver can very rarely be felt to be enlarged.[4] Gastric symptoms, and particularly nausea and vomiting, tend to accompany this condition.

Parotitis is a rare complication, but it is one whose appearance seriously increases the gravity of the prognosis. Most of the cases of Pneumonia in which it occurs prove fatal.[5] Grisolle states that its progress is very rapid, and that it tends to pass into suppuration or gangrene. In the former case, the pus may burrow deeply among the muscles of the neck, or may open into the external ear. The pus is, however, usually infiltrated, so that but little escapes on incision. It appears to be most common in advanced life. The only case in which I have met with it was in a girl aged fourteen.[6]

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[1] Dr. Parkes, Clinical Lecture, Med. Times and Gaz., 1860; i. 187.

[2] It occurred in 7 per cent. of Grisolle's cases, in less than 1 per cent. of 237 cases analysed by Roth, Würzb. Med. Zeitsch., i. Nos. 3 and 4. Cvostek (Canstatt's Jahresb. 1867) met with icterus in the proportion of 21 per cent. of 147 cases, and the mortality in these was 23·8 per cent. The average mortality of the whole number of these cases was 16·8 per cent.

[3] Out of 20 cases observed by Grisolle, 16 were associated with Pneumonia of the right lung. In these cases, however, the upper and lower lobes were affected with equal frequency.

[4] Andral reports a case (Clin. Méd. iii. p. 441, obs. lv.) of icterus accompanying Pneumonia, where the hepatic region was painful and resistant. The stools were natural, though all the tissues were stained with bile. Post-mortem, the liver was found softened, and of a deep red colour. The biliary passages were free; and bile could easily be expressed from the gall-bladder into the duodenum.

[5] Two such cases are related by Béhier, Conférences de Clinique Médicale.

[6] This case has already been referred to as an instance of Pneumonia passing into gangrene.

In rare cases an *inflammatory condition of the joints* occurs in the course of Pneumonia. Grisolle reports four such. In all these the joint affection was multiple, but it was not migratory. Three of these cases proved fatal. In the only one examined the joints contained pus, and Grisolle considers it probable that the affection was septic in its nature, since in all the fatal cases the lung was found in a state of suppuration. In one case, a patient of Dr. Reynolds, a man of dissipated habits, effusion came on in the knee-joint on the day after the crisis, attended with a slight rise of temperature. The Pneumonia resolved perfectly, but the swelling of the knee became chronic.[1]

VARIATIONS IN THE CLINICAL ASPECT OF ACUTE PNEUMONIA.

Many of these, depending on the severity of the coincident affection of the digestive or of the nervous system, have been already described. Three classes, however, deserve some mention, viz. Latent Pneumonia, the so-called Typhoid Pneumonia, and Pneumonia assuming an intermittent type.

LATENT PNEUMONIA.—The class of *Latent Pneumonias* is an ill-defined one, and in many cases in children the accompanying cerebral affection may mask the ordinary symptoms of the disease.

It is very rarely that in vigorous adults the inflammation of the lungs does not present characteristic clinical features, but in old people many of these are often absent. In cases also where Pneumonia is secondary to other diseases, the chief symptoms may be altogether wanting. In old people the disease may be only revealed by prostration, headache, and delirium, and none of the usual phenomena of invasion may be present. Cough also and expectoration may be entirely absent, or the latter may fail to present the characteristic rusty tint, and may be transparent and viscous or simply puriform. Subjective dyspnœa is also less frequent, though some acceleration of the respiration and the perversion of its normal ratio to the pulse rarely fail to be observed.

The flushed face is also less frequent in the aged than in adults, and the countenance is often pale, earthy, and sunken. The skin may be dry and hot, but it may fail to communicate to the hand the pungent feeling of heat sometimes described; or it may be relaxed and perspiring throughout. Fever is, however, almost always present, though seldom ranging so high as in adults and in children. Its presence is, however, a valuable indication for a careful investigation of the chest, since Pneumonia is one of the few febrile affections to which elderly people are liable. The disease, however, may be so en-

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[1] This patient was transferred to the surgical wards, and I am unable to trace his subsequent history.

tirely latent that its presence in a state of grey **hepatization may only** be revealed post mortem after a sudden and unexpected death.[1]

THE TYPHOID FORM OF PNEUMONIA is very common in elderly people, and might be described as a subvariety of the **Latent form.** Its occurrence, judging from my own experience, **must be rare in this** country, though some of the severer, and particularly of the fatal cases, tend to assume towards their close some of the characters described. Dr. Stokes, however, has found it more common in Dublin. It has also been described by Huxham **as occurring in** scorbutic patients, in whom it is often associated with dysentery attended by bloody stools. Huss remarks that it occasionally occurs sporadically, but only in those who have been exhausted by toil, want, or other depressing influences. It is very doubtful whether the reported epidemics of this character have been pure Pneumonia, or not rather typhoid fever.[2] Many of the cases in which Pneumonia occurs as a complication of other diseases tend to assume this type, but it may occasionally be met with as the primary disease. It may be described as a form of Pneumonia marked by intense prostration and by the signs of profound depression of the nervous centres. Its invasion is often gradual; the initial rigor may be slight or nil, an pain in the side may be absent or slight; the cough and sputa are often present at the outset, but the latter may be merely viscous or may present the characters of prune juice. Stupor, **alternating with** constant low muttering delirium, and associated with tremors and subsultus tendinum, with a fixed but vacant expression of countenance and with complete abolition of the senses of sight and hearing, also in some cases of the faculty of speech, are its most prominent features. The tongue is dry and brown, and sordes form on the teeth. Incontinence or retention of urine are sometimes observed. The pulse is small, but markedly accelerated. Sloughs may form on the most prominent parts. These symptoms may continue through the whole course of the disease, which usually ends fatally on the tenth or twelfth day, or later. The course in cases of recovery is commonly protracted, and resolution is very slow.

Wunderlich describes, as a variety of Pneumonia, a class of cases attended with early breaking down of lung-tissue (*Jäuchige Pneumonia*), which present a great resemblance to the typhoid form. T

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[1] Hourmann et Dechambre, Pneumonie des Vieillards (Arch. Gén. de Méd. 2e Sér. v 37). These authors state that of 19 cases of Pneumonia in old people uncomplicated disease of the heart or brain, 21 were latent. It is almost always latent when occurring in old people with cardiac or cerebral affections. See also Cruveilhier, Anat. Path. liv. xxxii.

[2] This would appear to be the case in the epidemic quoted by Grisolle, as described Torchet, at Noyers, Mém. Acad. Imp. 1838.

[4] Dr. Stokes, (loc. cit. p. 339) describes various forms: (1) As a complication "Enteritis, or Gastro-Enteritis;" (2) As a complication of true typhus; (3) Occurring in cases of bad erysipelas; (4) Occurring in cases of diffuse cellular inflammation; Occurring in cases of delirium tremens from excess; (6) As a consequence of phlebitis. (7) As apparently the sole disease.

sputa are fœtid and of a dirty colour. The fever is high, and prostration sets in early. Sweating is profuse, and there is a tendency to colliquative diarrhœa. Their course is protracted, and they tend to a fatal termination. When recovery takes place the fever subsides, and the sputa lose their fœtid odour and peculiar colour, and become simply purulent. I have seen one fatal case of this kind associated with dysentery, and with sloughs in the mucous membrane of the stomach.[1] Some forms of Pneumonia occurring secondarily to dilatation of the bronchi, are very prone to assume this character.

INTERMITTENT PNEUMONIA.—Among the inhabitants of malarial districts the symptoms of Pneumonia, and in particular the pyrexia, often assume an intermittent type.

The invasion is commonly attended with rigors, followed by pyrexia and sweating; but with these symptoms of ague the physical signs of Pneumonia may simultaneously make their appearance. In some cases, after the first twenty-four hours the fever ceases, and during the apyrexial period a marked improvement is said to take place in the physical signs : the dulness diminishes and the râles disappear, while the respiration over the affected part may be merely weak, or may in some cases retain the bronchial character. A second invasion, however, occurs with increased severity after twenty-four or forty-eight hours, with a return of the physical signs. The subsequent intermissions are less complete, but the pyrexia in such cases has always a distinctly remittent character, which may assume either the quotidian or tertian type; the cessation of the pneumonic signs in the early stages is, however, more complete in the latter than in the former variety. It is said that quinine, if given early, will cut the disease short; but if this is not effected, the Pneumonia tends to become double, and of a dangerous character.[2]

In some cases, however, of Pneumonia where there is no evidence of malarial infection, the type of the pneumonic pyrexia is distinctly intermittent, with apyrexial periods whose duration may vary from twelve to thirty-six hours. The remissions are attended with marked sweating, and also with an alleviation of the chief symptoms, though the physical signs usually remain unchanged during this period. The exacerbations are sometimes, but not always, attended by a return of the rigors which marked the primary invasion. This class of cases is rare, and the conditions determining their peculiarities are not fully explained. In some instances the exacerbations appear to be due to an irregular progress of the Pneumonia, but in others no determining cause, either of the remissions or of the return of the fever can be discovered.[3]

[1] Dr. Stokes (Cyc. Pract. Med. iii. art. "Gastritis") has also observed this form of Pneumonia associated with severe gastro-enteric disturbance. An instance of this form of Pneumonia is given by Dr. Laycock, "Fetid Bronchitis."

[2] See Morehead, Diseases of India, p. 349 et seq. Most of the other authorities on this subject will be found quoted in Grisolle's work.

[3] See Wunderlich, Die Eigenwärme im Krankheiten ; Thieme, Die Intermitterende Pneumonie, Diss., Jena, 1865 ; Griesinger, Virchow's Spec. Path. Therap., ii. p. 43.

PATHOLOGY.

A. MORBID ANATOMY.—The different anatomical changes which may be found in the course of acute sthenic Pneumonia have been ordinarily described under the terms of *Engorgement, Red Hepatization, Grey Hepatization, Suppuration,* and *Resolution.*

Dr. Stokes has, however, described a stage of *arterial injection* antecedent to that of engorgement, and characterised by a brighter colour and by dryness of the pulmonary tissue. Opportunities for observing this condition are extremely rare, and its very existence has been called in question by Rokitansky and by Skoda. There is, however, reason to believe in the probability that such a state may precede the subsequent changes of the inflammatory period, and the auscultatory signs of harsh respiration, which have been described by Dr. Stokes as attending it, have been recognised by many and different authors.[1]

(1) *The stage of Engorgement* is characterised by intense congestion of the pulmonary vessels and by commencing œdema of the lung.

The tissue is of a deep reddish-purple tint. It is heavier than natural, and has lost some of its resistance and elasticity. It pits on pressure, and is more easily torn than a healthy lung. On section a large amount of blood-stained serosity escapes from the cut surface, and in the earlier stages this is frothy from the admixture of air. During this period the tissue is still crepitant, and floats in water to a degree corresponding with the extent to which the condition has advanced. Under the microscope, the capillaries of the pulmonary artery are found to be loaded with blood. The epithelial cells of the air-vesicles are seen to be enlarged and granular, and occasionally they exhibit a commencing division of their nuclei: some exudation-corpuscles may also be seen in the alveoli, mingled with red blood-corpuscles which have escaped from the capillaries.

The question of the vessels chiefly concerned in the pneumonic process has been largely discussed without any definite settlement having been arrived at. It has been maintained by some that the inflammatory changes are mainly dependent on the bronchial artery as the nutritive vessel of the lungs,[2] and Virchow's observations have shown that the most typical pneumonic changes may ensue in parts of these organs whose supply from the pulmonary artery has been completely arrested by the occlusion of branches of this vessel.[3] It is by

In none of the reported cases of this condition with which I am acquainted has the condition of the spleen been mentioned.

[1] The reality of its existence must, however, in part depend on the question of the increased arterial supply from the bronchial vessels, since congestion of the capillaries of the pulmonary artery does not give this tint.

[2] This question, according to Virchow, was first raised by Boerhaave. (See Van Swieten, Comm. in Aph. Boerhaave, ii. 712.) It has also been ably discussed by Dr. Morehead, Dis. of India, ii. 311.

[3] Ges. Abhand. p. 369 et seq. Dr. Waters (Dis. of Chest, p. 30) believes that the pulmonary artery is exclusively distributed to the walls of the air-vesicles. Since, however, it has been shown that some of the products of inflammation may escape by the

no means, however, certain that the nutrition of the lung is exclusively conducted by the bronchial artery; and it is not at all improbable that the branches of the pulmonary artery, whose participation in the process of congestion so vastly exceeds that of the bronchial capillaries, may have no inconsiderable share in the exudative processes which distinguish the condition of hepatization.

(2) *Red Hepatization* is the term generally adopted for the appearance observed in the second stage. In it the lung has become solid; it sinks in water, and the section is that of a solid tissue. It is firm, as if the lung had been artificially injected with size from the bronchi; but it has lost its elasticity and resistance, it tears easily, and breaks down into a pulp under pressure. Its section is less livid than that of a simply congested lung, and is of a dull, reddish-brown tint (sometimes likened, but not very exactly, to mahogany), which, however, becomes brighter after a short exposure to the atmosphere. It is also opaque, and has lost the glistening transparency of ordinary pulmonary tissue.[1] The colour is not absolutely uniform, but mingled with the reddened tint is a greyish appearance, as if Chinese white had been mixed with the colouring matter. Very little serosity exudes on section, but a dirty, rusty-looking, reddish fluid with a certain degree of viscidity may be expressed or scraped from the surface. A characteristic appearance of the section in the Pneumonia of adults is the granular look which it presents, and which is still more distinct when the tissue is torn. The granulations are small and uniform; they give the torn surface the appearance seen on the exterior of a nutmeg, and they may easily be separated on scraping the tissue. This granular appearance is less distinct in children, and varies also in degree according to the amount of œdema present.[2]

During this stage the interlobar septa, and even the larger bronchial vessels, are still distinct, and participate but little in the inflammatory changes, but the latter are sometimes found filled with solid exudation-matter. The vesicular character of the lung is, however, entirely destroyed, being replaced by the granular look just described.

The tissue is greatly increased in weight, and, according to Gendrin, its specific gravity when compared with that of healthy lung may be as 1·15 or 1·9 to 1.

The lung is expanded by the exudation present to the fullest capacity of its normal dimensions. It is possible also that it may somewhat exceed this. The possibility of its thus retaining the impress of the ribs has been largely discussed; but it has been definitively settled in the affirmative.

The pleura almost invariably participates in the inflammatory

veins, it is possible that this may explain such cases as those described by Virchow; though some doubt still remains as to whether the bronchial arteries may not participate in the process more than Dr. Waters' suggestions would lead him to believe.

[1] It is to be remarked that this translucency is preserved in conditions of collapse, and that the dead opacity of appearance is one of the best characteristics of the pneumonic process.

[2] See Appendix C.

changes when the part affected is superficial. It loses its normal translucency and becomes opaque, and it is generally covered with a layer of fibrinous exudation.

When the stage of red hepatization has lasted some days, its colour becomes paler and whiter. This is due to individual granulations becoming whiter in aspect, either singly, or in groups scattered through the surrounding reddened tissue ; and this change produces a mottled look in the inflamed part. Coincidently with this change of colour there is a gradually increasing loss of the solidity of the affected tissue: the exudation liquefies, and more fluid can be expressed from the cut surface, and the state may gradually pass into that of grey hepatization, though it is very questionable whether perfect resolution does not often take place without the latter being fully attained.

The two conditions are, however, frequently found intermingled, and the lung then acquires a marbled appearance which, as Laennec remarked, may closely resemble some forms of granite.

(3) *Grey Hepatization.*—In this condition the cut surface of the affected part is of a uniform grey tint, generally presenting, however, a somewhat greenish or olive tinge. The redness of the preceding stage has disappeared entirely, and the granular character has become less distinct. The tissue has lost its firmness and has become soft and pulpy, and allows a dirty-looking, puriform, grey fluid to be abundantly exuded, both on scraping and on pressure. Sometimes a further stage of softening is reached, though this is, comparatively speaking, very rarely observed. Many minor variations of appearance are presented in this state, which usually is found in persons of bad constitution or in cases where Pneumonia is secondary to other diseases. The difference in the appearances observed depend, however, for the most part, on the greater or less amount of œdema present, and in the comparative indistinctness of the granulations. In some instances these are entirely absent, and the tissue is uniform, smooth, and glistening. Under these circumstances a large amount of serum may escape on pressure, containing but few solid elements, and not presenting, therefore, the milky, puriform detritus usually observed. Such conditions are not uncommon in cases of Pneumonia proving fatal in the course of Bright's disease, when attendant œdema of the lung complicates the inflammatory process. In some instances also this condition appears capable of remaining for some time in a chronic state, when it may form one of the stages of transition between acute and chronic Pneumonia. This is particularly the case in some forms of phthisis, but it is also seen independently of the complication with tubercles. The consolidated lung still retains the grey marbled appearance, and some serosity may escape on pressure, but the tissue gradually acquires a more resisting character, and does not break down easily into detritus.[1]

[1] I doubt much whether cheesy changes in the exudation, as described by Niemeyer, are commonly observed in this form of Pneumonia, independently of tubercular formations in the walls of the air-vesicles. Such an event may be possible, but I believe that cheesy masses commonly found in such lungs in phthisical patients are usually, though

(4) *Suppuration of the Lung.*—In this state the lung presents a yellower appearance than that seen in the grey hepatization. The granular character is lost, and a diffluent puriform fluid exudes from the cut surface. The whole tissue of the lung is softened and pulpy, and breaks down with the greatest facility under very slight pressure, and it may thus give rise to the false impression that an abscess has been formed. The condition is not, however, specifically distinguished from either of those last named, in respect of the changes in the pulmonary tissue, since pus-cells are present in all stages of the pneumonic process; and the greater degree of softness and the changes of colour observed in the so-called grey hepatization and suppuration of the lung are only due to the increasing anæmia caused by the pressure of the accumulated products of inflammation in the interior of the air-vesicles, and by the progressive degrees of fatty degeneration in the cell-forms thus produced: while the gradual softening is attributable to the liquefaction of the previously solidified exudation.[1]

(5) During *the stage of Resolution* the liquefied exudation matter, and the cell-forms which have degenerated and broken down, are gradually absorbed. The expectoration is often in such cases so insignificant as by no means to account for the elimination in this manner of these products, and the greater part must necessarily be removed by absorption. Opportunities for the observation of lungs in this condition are rare. I once found, three weeks after the physical signs had disappeared, a considerable amount of œdema remaining in the affected parts, together with a marked loss of elasticity of the tissue.[2]

It is a matter of some interest that Pneumonia has been described as a disease of intra-uterine life. F. Weber[3] mentions it as existing in two forms, a white hepatization and a red. The former, however, is now generally considered to be a syphilitic affection. The red hepatization of intra-uterine life occurs as a lobar Pneumonia. It is most commonly met with during epidemics of puerperal fever, which Weber believes may produce blood-poisoning in the mother before delivery.[4] The lung is very much gorged with blood, and is softer than in the ordinary form of red hepatization, though resembling the

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not invariably, the result of a secondary tubercular growth. The discussion of this question, however, belongs to that of Phthisis, and cannot be entered upon here.

[1] It has been repeatedly affirmed that this condition is not a true " suppuration of the lung " as stated by Gluge, and certainly it does not specifically differ from the previous stages, since pus-cells are produced throughout the whole pneumonic process. The question is one of terms rather than of a reality, but as the contents of the air-vesicles are more purely puriform than in the earlier stages, there appears to me to be no objection to retaining the expression.

[2] An accident prevented my making a microscopic examination of this lung. Similar conditions have been described by Laennec and Grisolle. Laennec's description of the process is very minute, and subdivided according to the different stages. It would appear, however, doubtful whether these can be so perfectly defined as was attempted by him.

[3] Path. Anat. des Neugeb. und Säuglinge, ii. 41 et seq.

[4] Forster (Handb. der Path. Anat., 2d Ed. ii. 248) says that he has met with this change under similar circumstances.

Pneumonia found in some conditions of blood dyscrasia. The disease usually proves fatal within a few hours after birth.

The microscopical examination of a pneumonic lung[1] is at once sufficient to show that the inflammatory products are almost entirely accumulated in the interior of the air-vesicles. This is seen in Fig. A ($\times$ 100 diam.); and the same appearance persists throughout
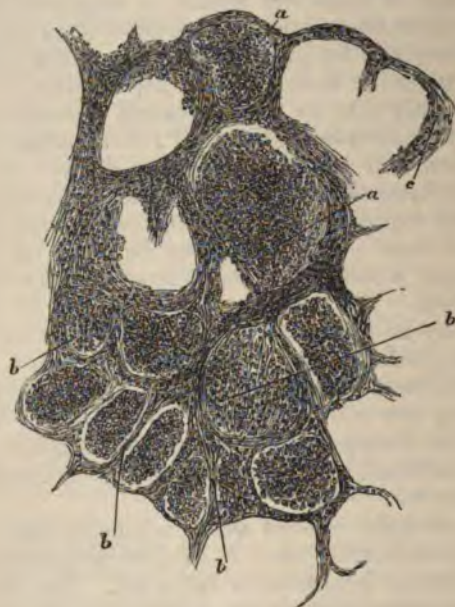
Fig. A.

all the stages of the process, including that of grey hepatization, in which, as originally remarked by Gluge,[2] the elastic fibres are still distinct. The walls of the vesicles are, however, somewhat swollen, but this is almost entirely owing to the congestion of the capillaries, and there is an entire absence of any intersticial growth or exudative process within or external to them. In some parts, *b b*, in hardened preparations, the contained masses of cells separate from the walls of the air-vesicles, leaving the latter intact.

When examined with a higher power (Figs. B and C, $\times$ 700), the alveoli are seen to be occupied by a considerable variety of cell-forms held together by a tenacious material, and mingled with a number of free red blood-corpuscles (Fig. B, *b*). The amount of these latter, however, varies greatly, but in some instances it may be so excessive as to

[1] On this subject see also Dr. Da Costa, "Amer. Journ. Microscop. Science," 1855; and Rindfleisch, "Lehrb. der Path. Gewebelehre."
[2] Anat. Microscop. 1838.

form a large proportion of the material filling the alveoli. In the earlier stages of the process, the epithelial cells of the alveoli and

Fig. B.

smaller bronchioles are seen in different stages of transformation and proliferation. They are greatly enlarged, measuring from $\frac{1}{1500}$ to
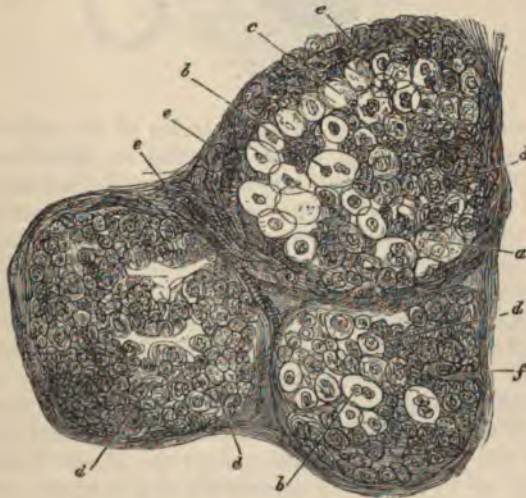
Fig. C.

$\frac{1}{2000}$ or $\frac{1}{2500}$ of an inch in diameter. They tend to assume the round form, but some (Fig. D, *a b d*) are at times irregular in shape. They are for the most part very granular.[1]

[1] In Figs. B and C, the preparations from which the drawings were made were put up in Canada balsam or Damara gum, and the antecedent modes of preparation (immersion in turpentine and chloroform) dissolved out the fat granules.

In the early stages they are cloudy and opaque, but they clear with acetic acid, showing that they contain an excess of fibrinous matter; but as the process advances, the granular character is mainly due to the accumulation of fat drops in their interior. The nuclei in these cells are sometimes single, and show a distinct nucleolus (Fig. B, *a;* Fig. D, *a c c*); but in the majority of instances the nuclei may be seen in all stages of multiplication and division (Fig. C, *b b;* Fig. D, *b e d*), until several

FIG. D.

nuclei are found accumulated in the interior of the cells (Fig. D, *f*). Large cells may, however, at times be found in other conditions in which the nucleus has disappeared, and the cell may only present a clear hyaline cavity in its interior, which gradually increases in size until, in some instances, only a narrow margin of the granular cell contents is seen surrounding the central space (Fig. C, *c;* Fig. D, *k k*). Together with these there are seen various forms of pyoid cells, some of which present one, and others two nuclei or more (Fig. D, *f*). They are smaller than the foregoing, and average from $\frac{1}{2000}$ to $\frac{1}{2500}$ of an inch in diameter; the nuclei vary in size from $\frac{1}{5000}$ to $\frac{1}{8000}$ of an inch. Many round cells are also seen in which no nucleus is apparent (Fig. D, *h*). Some of these correspond in appearance with that presented by the nuclei of the larger cells; others bear the closest resemblance to lymphoid cells or to the white corpuscles of the blood; others again are larger than these (Fig. D, *i*). The whole of these cells are finely or coarsely granular, the granules being mainly of a fatty nature. They are often stained by imbibed hematine, and in the later stages pigment granules tend to accumulate in increasing numbers in their interior. They are seen in Figs. B, C, to be irregularly scattered among the larger epithelial cells.

As the process advances, the granule cells become more numerous, and the epithelial cells in great measure disappear. This is

due to the fatty disintegration of the latter, which may be seen in all stages of this change, large tracts being filled with coarser granule cells, and with the compound granular bodies of Gluge. They break down and their nuclei are set free, until the interiors of the alveoli are almost entirely occupied by the smaller-sized round

Fig. E.

nucleated and non-nucleated cells (Fig. E), in which large quantities of fat granules become accumulated. These appearances are most common when the stage of grey hepatization is reached; but similar conditions are often found in parts which to the naked eye still present the aspect of red hepatization. In the earlier periods the cells are agglutinated together by a material of a cohesive nature, which is usually considered to be fibrin, but of the nature of which no very precise chemical proof has been afforded;[1] but it may sometimes present a fine network like that seen in whipped fibrin from the blood. Its cohesive nature is, however, distinctly seen in the fact that the granules may be scraped or washed out entire from the cut surface, and these not infrequently present the form of casts of the smaller bronchi and infundibula, and consist of masses of the cells now described. If a section of the lung in this state be carefully washed over with a camel's-hair pencil, cells are seen still remaining between and imbedded among the elastic fibres of the alveoli, mingled

[1] In the earlier stages of the pneumonic process, during the period of engorgement, the air-vesicles are loaded with a clear but very tenacious fluid, which, however, becomes cloudy on the addition of acetic acid. During the height of the consolidation acetic acid effects a partial clearing of the effused material, while during the stage of liquefaction the qualities of this fluid in respect to the reaction with acetic acid revert to the first stage. (Rindfleisch, Lehrbuch der Path. Gewebelehre, 363.)

with an adventitious network of a fibrinous nature (Fig. F). These fibres have not, however, the definite outline and the regular arrangement seen in the process of growth which characterises tubercular formation, and, though in section some cells are seen irregularly scattered over the walls of the alveoli, no intersticial growth appears to take place in these during the process of acute Pneumonia. In the later stages of the process the material holding the cells together loses much of its cohesive properties, and becomes more fluid,
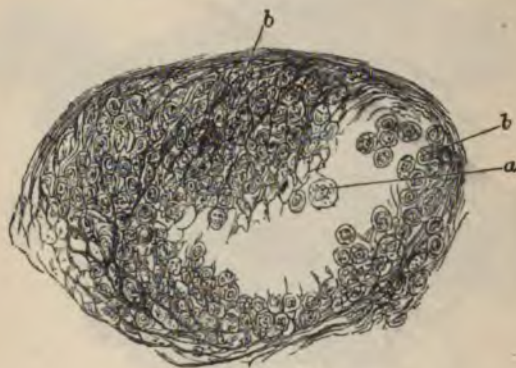
FIG. F.

and, together with the cell-forms observed, great numbers of free oil globules and of granules of protein matter become apparent. In this stage, scrapings of the tissue yield only cells and a tenacious fluid, and neither the granules nor the casts of the bronchi can be separated entire.

The full discussion of the pathology of these processes, involving as it does the whole question of the nature of the changes of tissues in inflammation, can necessarily be only briefly dwelt upon here. The points of greatest interest in relation to it are those regarding the nature of the exudation process, and the origin of the cells which are produced in such excess in the interior of the air-vesicles. Until the publication of Cohnheim's researches, the opinion generally received was that of Virchow, that the coagulable material was derived from blood-plasma, changed during its passage through the inflamed tissues, and that the cell-forms found were the result of increased growth from pre-existing tissues. Cohnheim's[1] statement, that the so-called pus-cells in inflammatory processes consist chiefly of the white corpuscles of the blood which have passed through the walls of the blood-vessels, has been absolutely adopted by Professor Axel Key[2] in respect to Pneumonia, though here the kind of proof obtainable in the mesentery

[1] See Appendix D.
[2] Hygeia, 1868, p. 530. Translated by Dr. W. Moore (Med. Times and Gazette, 1869, 452).

and in the tongue of the frog is necessarily wanting. That such a passage takes place in these parts in the frog can be easily verified, but that this migration of the white corpuscles is the sole source of the vast increase of cells found in inflamed parts appears to me inconsistent with facts. I believe that all the cells and nuclear elements (centres of nutrition) of a tissue participate in the inflammatory process, and multiply in number by division.

The illustrations which I have given of these processes during Pneumonia are, I think, sufficient to confirm this proposition, and the same fact may be abundantly seen in the frog's mesentery, where, in parts when no escape of corpuscles from the blood-vessels is taking place, and even before this process has commenced, a great increase of the nuclei in the tissue may be observed without a single corpuscle having migrated from elsewhere into such parts.

The tendency of all irritative growth is to approximate to what may be considered as the primary or lymphoid cell-forms, and hence " pus-cells," which in some cases are undistinguishable from the white corpuscles of the blood, are produced wherever rapid growth of this nature occurs. I believe, therefore, that while a number of the cells in the pulmonary alveoli may be those which have escaped from the blood-vessels of this part, another series are produced in the manner above described, and which in their final stages are undistinguishable from the former.[1]

With respect to the exudation, it may be held, when spontaneously coagulable, to consist mainly of the blood-plasma; but the condition of this product in the early stages of Pneumonia suggests at least a doubt whether it is merely a transudation, or not rather, as Virchow has taught, that it owes some of its properties or transformations which it has undergone during its passage through the inflamed tissues.

The terminations of acute Pneumonia in *abscess* and *gangrene* are very rare.

Abscess[2] is probably the rarer of these. It is due directly to the

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[1] That a local production of white corpuscles takes place either in the lymphatics or in the blood-vessels (and probably in both) of an inflamed part, is, I think, probable from the great increase of these in the blood generally. If this were not the case their exit at the seat of inflammation ought largely to diminish their relative number in the systemic blood, whereas precisely the reverse is observed.

[2] Huss estimates its frequency as once in 50 or 60 cases; Laennec only saw five instances, but he probably overrated their frequency as diagnosed by physical signs. Chomel met with it three times; Louis, Andral, and Grisolle have each observed only one instance. Morehead (loc. cit.) in 189 cases only found five instances. Of twenty-five cases collected by Grisolle from different sources, eight occurred above ætat. 70, twelve above ætat. 50, and three above ætat. 45. According to the statements of Barthez and Rilliet, and of all other authors, abscesses are very rare in the primary Pneumonia of childhood, contrasting in this respect with the effects of Broncho-pneumonia. They appear to be proportionately more frequent in Pneumonia of the upper than in that of the lower lobes, especially when the relative liability of these parts of the lung to the primary disease is considered. Multiple abscesses are most commonly the result of pyæmia, and have been described under this head. (See Vol. I. of this work.) Rare instances may be found of abscesses in the lung caused by those formed in the different organs of the abdominal cavity perforating the diaphragm. Several cases of this kind have been collected by Dr. Stokes. (See also Ulcer of Stomach, Vol. II.) Foreign bodies entering the lung are also an occasional cause.

breaking down of the lung-tissue, and it is most commonly found in parts which are the seat of grey hepatization. The size of such abscesses varies from that of a bean or a pea to a cavity of some inches in diameter.[1] They may in rare instances give rise to pneumothorax, and a case is reported of an opening being effected into the pericardium.[2] They are sometimes found surrounded by a thickened wall of false membrane, but more commonly they merely form irregular excavations in the softened tissue which may hang in irregular necrotising rags in their interior.

Gangrene has been already stated to be rare in acute primary Pneumonia, but its occasional occurrence seems to be indisputable.[3] It may invade considerable tracts of tissue. The distinction between some forms of grey hepatization and true gangrene is not always very sharply defined. The former may be found rapidly breaking down into a pulpy detritus of a dirty and blackened appearance, but wanting the characteristic odour of a gangrenous lung. Such states appear to occur most commonly in persons of bad constitution or under peculiar conditions of blood-poisoning.[4] This state corresponds with the Ichorous (*Jäuchige*) Pneumonia of Wunderlich, to which allusion has been already made. The condition is usually accompanied by intense typhoid prostration, and the Pneumonia only appears to be part of a constitutional state which is of the extremest gravity.

When true gangrene takes place, the part affected is dark and stinking, and is commonly reduced to a pulpy débris. The gangrenous fragments are not infrequently found floating in a pseudo-cavity amid fœtid putrilage. Huss attributes its origin to thrombosis occurring in the branches of the pulmonary artery—a view originally entertained by Carswell;[5] but in some instances it may be due to the directly destructive effect of the inflammatory process destroying the vitality of the tissue, or to an arrest of the circulation by the excessive accumulation of its products in the interior of the air-vesicles. The portions affected are commonly surrounded by tissue in a state of grey hepatization.

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[1] A remarkable case of the cicatrisation of an abscess is reported by Dr. Stokes. Laennec describes one involving the greater part of the middle and lower lobes.

[2] Béclard, Bull. Soc. Anat. 1863, p. 356. (Grisolle.)

[3] Andral, Clin. Méd., obs. vol. iii. 63 and 68. Willigk (Prager Vierteljahresch. xxxviii. p. 13) found gangrene in 52 out of 583 post-mortems of Pneumonia = 3·3 per cent. It occurred in 3·6 per cent. of the males, and 2·9 per cent. of the females. Huss, in 2,166 cases of Pneumonia, met with only 12 instances : all were in males, aged from 35 to 55, and all the cases were in patients of exhausted constitutions. Dr. West (loc. cit. p. 318) says that "the lung in childhood shows a much greater tendency to pass into a state of gangrene than in adult age." This tendency, however, is not seen in the acute Pneumonia of children, and instances of such an occurrence only occur singly in the works of different authors. (See Steffen, loc. cit.) Ziemssen (loc. cit.) met with only one case out of 201 instances of primary Pneumonia in children.

[4] See a case by Bamberger (Deutsche Klinik, 1850, 115) of Pneumonia of this nature passing into abscess. It occurred five days after parturition. A case is reported by Dr. Laycock (Fetid Bronchitis, p. 27) of acute gangrenous Pneumonia destroying nearly the whole of one lung, and proving fatal within a month. Tubercles were present in this case.

[5] Illust. Elem. Forms of Disease, art. "Mortification."

Site.—The most frequent *seat* of acute primary Pneumonia is in the lower lobe of the right lung.

The excess of frequency of the affection of the right lung, independently of the seat of the disease, over that of the left, is variously stated by different observers [1] in the proportions of 5 to 3 or 7 to 4. This predominance of the right side over the left exists from the earliest infancy, but diminishes somewhat with advancing age.[2] The same relative proportions obtain equally for both sexes.

The lower lobe is affected more frequently than the upper in both lungs collectively in the proportion of about 3 to 2.[3] The proportion remains nearly the same for childhood[4] and adult age; but in more advanced periods of life there is a greater tendency to invasion of the upper lobes. This proportion, however, according to Dr. Stokes, varies from year to year, and it appears also to be sometimes influenced by epidemic causes, rendering the upper lobe more liable to suffer than the lower.

In relation to the lung affected, Pneumonia of the upper lobe is singularly more common in the right than in the left lung,[5] and with a relative frequency which is greatly in excess of the proportion observed in the affections of the two sides, when considered independently of the locality of the inflammation.

The middle lobe of the right lung is still less frequently affected. Dr. Walshe states that Pneumonia having this site is usually either the result of endocarditis, or that it depends on blood-poisoning.

Double Pneumonia is comparatively much less frequent than the unilateral affection, except in the case of Broncho-pneumonia.[6] The liability of old people and children to this form of the disease has, however, led to an exaggeration of the frequency with which both lungs may suffer. It is comparatively rare that both lungs are

[1] Huss, of 2,616 cases, found 53 per cent. in the right lung, 32 per cent. in the left, and double Pneumonia in 15 per cent. Grisolle, in 1,430 cases collected from various authors, found 742 cases in the right lung, 426 in the left, and double Pneumonia in 262. The latter number he regards as doubtful.

[2] Hourmann and Dechambre found in old people the proportionate frequency of Pneumonia of the right lung to that of the left, as 34 to 27.

[3] This is Andral's statement. Wunderlich, in a calculation of 660 cases, from different authors, gives the following numbers:—Lower lobes, 397 cases; upper lobes, 180 cases; affection of a whole lung, 83 cases. Grisolle, from a calculation of 264 cases, gives the proportions as—lower lobe, 133; upper, 101; middle lobe, 30 cases.

[4] Barthez and Rilliet (i. 516) found in 122 cases: Upper lobe 42; lower lobe, 65; affection of the whole lung, 3; double Pneumonia, 12 cases.

[5] Grisolle states that Pneumonia of the upper lobe in the right lung is two and a half times more common than in the upper lobe of the left. The observations of other authorities show a still more striking difference. Barth states the relative frequency of Pneumonia of the upper lobe of the right lung to that of the left as 18 to 1; Briquet as 18 to 4; Barthez and Rilliet as 9 to 1. Ziemssen, in 234 cases, gives the following numbers:—Right side collectively, 126 cases, upper lobe, 57; lower lobe, 55; middle lobe, 14. Left side collectively, 106 cases; upper lobe, 27; lower lobe, 79.

[6] The proportion of 15 per cent. given by Huss represents nearly the average frequency of its occurrence, but the smaller numbers of some observers show different ratios. Grisolle states its frequency to be 11 per cent. The Vienna returns for 1860 place it at 5 per cent. Barth, in 125 cases, at 6 per cent. Willigk, on the other hand, loc. cit., found double Pneumonia in 50 per cent.

attacked simultaneously, and the invasion of one is commonly secondary in point of time to that of the other. No difference appears to exist in the relative liability of either side to be followed by the attack in the opposite lung.

The Mode of progressive Extension of the disease is usually direct from the site first implicated. Exceptions to this, however, occur when the opposite side suffers subsequently, and also sometimes when the upper lobe is invaded after the disease has commenced in the lower lobe of the same side. It is also not very uncommon in fatal cases to find disseminated nodules of pneumonic change, varying in size from a hazel-nut to a walnut, scattered irregularly around, and sometimes at a considerable distance from the larger mass, and separated from it by apparently sound, or sometimes by unduly hyperæmic tissue.

The Rate of Evolution of the different stages of the process appears to be very variable. A lung may remain in a condition of red hepatization during eight or ten days, or even for some weeks; while in others the condition of grey hepatization may be found as early as the fourth or fifth day.[1] Both Laennec and Huss consider the stage of engorgement to last from one to three days; whether it can persist longer than this without producing some consolidation of lung is, however, doubtful. It is not uncommon for twenty-four hours to elapse before the physical signs of consolidation become apparent; but the duration of the stage of engorgement may be so short that a large tract of lung may be consolidated within a few hours after the first rigor.[2] The duration of the stage of red hepatization was stated by Laennec to vary from one to three days, and of suppuration from two to six days. Huss reckons the former as lasting from five to seven days. The periods of resolution have been already referred to.

The terminations in abscess or gangrene usually occur at later periods; but even the latter, as has been already stated, may be found very early in the course of the disease.

THE PATHOGENESIS of acute primary Pneumonia is involved in considerable obscurity, and has been the subject of much discussion.[3] Two opposite theories have been advanced respecting its origin, both of which are supported by certain facts and are opposed by others.

These theories may be briefly stated in the following terms:—

(1) That Pneumonia is a "specific" fever, of which the disease in the lung is only a local effect.

[1] I have seen this in a child under æt. 1, when the disease began acutely. Laennec, (Forbes' Trans. p. 206), states that the stage of purulent infiltration may be reached in thirty-six hours.

[2] Dr. Stokes, loc. cit., p. 120, remarks that in some instances of typhoid pneumonia there is no evidence of an antecedent stage of engorgement, but that the lung may become solid without any crepitant râle preceding this change. He further observes, however, that this rapidity of progress is not common in the sthenic forms of the disease.

[3] See especially on this point a clinical lecture by Dr. Parkes, Medical Times and Gazette, 1860, i. 187.

(2) That it is a purely local disease, of which the pyrexial and other phenomena observed are only the immediate consequences.

The second hypothesis, as such, appears to be scarcely a tenable one, and even the first appears to require some modification.

The arguments in favour of the first hypothesis are mainly derived from the comparative rarity of discoverable causes for the origin of Pneumonia,[1] and from the suddenness of the crisis while the inflammation is still at its height.

The question of the mode of origin of the disease has been already considered under the head of etiology. It appears, however, deserving of remark that the theory of a "specific" cause can scarcely be maintained for Pneumonia in the same sense as that in which the term is employed for the contagious pyrexial diseases. The causes of Pneumonia are manifold, and the disease may originate under such diverse conditions, that it seems impossible to attribute it to any single blood-poison.

On the other hand, the most probable hypothesis to explain its origin is that of an altered composition of, or the existence of some morbid material in, the blood, which from its special qualities may affect a particular organ, or, as is more probable, may, under local predisposing causes, excite inflammation in that part of the system which in any given individual is the most liable to suffer as a *locus minoris resistentiæ.*

It is not improbable that some of the antecedent symptoms, the malaise, the pains in the limbs, the headache, and the slight jaundice occasionally observed, may be due to the blood alteration; but it must be remembered, that in a not inconsiderable proportion of cases, the outbreak of the pneumonic fever is sudden, without being preceded by any of these prodromata. The nature of the alterations in the blood capable of producing the disease are, like those of all other spontaneous inflammations, entirely unknown; and the hypothesis of an antecedent condition of hyperinosis advanced by Naumann[2] seems to be disproved by some of Zimmermann's analyses. It would appear indeed from these that the excess of fibrine observed in the blood of pneumonic patients is almost entirely a secondary phenomenon, and that it is, as Virchow affirms, a consequence and not a cause of the inflammatory process in the lung.[3]

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[1] It has already been stated (see Etiology) that experimental attempts at the production of a disease resembling acute primary Pneumonia by direct irritation of the lung, have invariably failed.

[2] Ergebnisse und Studien aus der Med. Clin. zu Bonn, 1858. Naumann has afforded no direct proof of an increase of fibrine in the blood antecedent to an attack of pneumonia. He says, however, that symptoms resembling the prodromata of pneumonia are sometimes associated with hyperinosis.

[3] Thus, in "Prager Vierteljahresch." 1852, vol. xxxv., in a patient bled, after signs of the stage of engorgement had lasted five days, the blood contained only 1·13 per 1,000 of fibrine. In a venesection practised 36 hours later, and when signs of consolidation had supervened, the fibrine amounted to 4·41 per 1,000, and in a third venesection practised after 6 days it had risen to 7·16 per 1,000. Zimmermann argues further, that this rapidly

The theory of hyperinosis as a cause of Pneumonia has also but little support in the diseases with which it is commonly associated, for though it is a not uncommon complication of acute rheumatism, in which this condition of blood is present, it also occurs in other diseases when the amount of fibrine is below the normal standard,[1] and in some of these, as in typhoid fever, the supervention of pneumonia increases the proportion of fibrine in the blood.[2]

It would appear from the consideration of the various diseases with which Pneumonia may be associated, that many, and probably different, blood-poisons may have the power of exciting inflammation of the lungs.

That the lungs should be especially liable to become affected by causes of this nature cannot be regarded as extraordinary, when we consider the importance of their functions as purifying agents of the blood. Nor does it seem improbable, from the complexity of the lymphatic structures which they contain, that other changes in the composition of the blood, in addition to its mere aëration, may be accomplished by their means, though of the nature of these changes we are as yet ignorant.

The lung, from its embryological development and anatomical characters, is closely allied to the glandular organs, and it is on these that blood-poisons produce their most marked effects. It is further to be noted that other organs of this class are not unfrequently simultaneously

increasing quantity of fibrine in the blood, in the later stages of Pneumonia, is not due to venesection, for in other cases bled *for the first time* on the third, fourth, and eighth days respectively the fibrine amounted to 7·2, 8·0, 9·1, and 9·6 per 1,000. Also in "Analyse des Blutes," p. 370, he has, as the result of more extended observations, found that in eight cases of commencing Pneumonia the blood contained either a normal or a less than normal amount of fibrine. In eight other cases where venesection was practised within the first 24 hours, the proportion of fibrine was between 3 and 7·5 per 1,000, and in eight other cases, the blood in the second 24 hours of the disease contained fibrine varying from 3 to 7·5 per 1,000. Zimmermann's method of analysis leads him to estimate the amount of fibrine in the healthy blood as lower than that given by many observers; but this only adds strength to his estimate of the proportion observed in inflammatory diseases. He places it at 1·689 per 1,000 (loc. cit. p. 17), while Becquerel and Rodier estimate it at 2·5, and Andral and Gavarret at 3 per 1,000. The increase of fibrine in the blood during the progress of Pneumonia is abundantly confirmed by other observers. Thus Andral (Ess. Hæm. Path. p. 87), in 90 cases, found in

7	cases fibrine	4—5	per 1,000
17	,,	5—6	,,
19	,,	6—7	,,
15	,,	7—8	,,
17	,,	8—9	,,
9	,,	9—10	,,
6	,,	10 and upwards.	

The largest amount in Pneumonia recorded by Andral is 10·5 per 1,000, and Zimmermann (loc. cit. p. 13) also found 10 per 1,000.

[1] I have already advanced reasons for doubting whether the Pneumonia which is secondary to many of these diseases differs essentially in anatomical characters from that of the acute primary disease. The firmness of the exudation varies in degree, but this may be influenced by the nature of the disease to which it is secondary.

[2] Andral, Ess. Hæm. Path. 17.

affected. The frequent association of albuminuria with Pneumonia can scarcely be regarded as a mere accidental complication, and it is by no means improbable that the kidneys are, under these circumstances, implicated by the same cause as the lung. Other glands also occasionally suffer, as the parotid : gastro-duodenal catarrh and some degree of affection of the liver are also frequent complications. In addition to these, the serous membranes tend also to become implicated as part of the primary disease, and when these relations of Pneumonia are regarded as a whole, it appears that those organs are most likely to suffer which are most commonly affected by recognisable conditions of blood-poisoning. The fact that cases of Pneumonia presenting these complications are more severe and dangerous than the simple disease, would also tend to show a greater intensity of the primary cause, for their mortality is disproportioned to what might be expected (particularly in cases where parotitis is present) from the mere existence of these inflammations, if regarded as purely local disorders. The argument is still further strengthened by the profuse sweating which often attends Pneumonia, and also by the frequent co-existence of herpes, which is so commonly associated with disordered blood-states.

In some cases Pneumonia indeed is known to be caused by recognisable conditions of this nature, as by septicæmia, but in the case of the acute primary disease it is most probable that the poison is one engendered within the system. In the cases where a discoverable cause exists, such as a chill, it is probably due to retained products of secretion injuriously affecting the composition of the blood. It is also not improbable that Pneumonia secondary to uræmic poisoning may have a similar origin ; while in the cases where no discoverable cause exists, we only stand, as has been already remarked, in the same position with respect to Pneumonia as we do to other idiopathic local inflammations.

Whether the blood-poison is eliminated by the exudation process must remain a matter of hypothesis, though the sudden cessation of the pyrexia when this stage has advanced to a certain degree would appear to lend some support to this view, and particularly when we remember the analogy, and even the various phases of transition, which exist between exudative and secretory processes.

The sudden outbreak of the pyrexia occurring simultaneously with the supervention of the inflammatory changes in the lung would, however, appear to show that the implication of the nervous system indicated by the fever is largely due to the alteration of the composition of the blood produced by the local process. We have no evidence of any distinct alteration antecedent to this, and much that a large proportion of the subsequent changes in the blood are due mainly to this cause.

All local inflammations produce in this respect similar results,[1] and it is interesting to remark that the pyrexia following a purely traumatic

[1] See Andral, loc. cit. ; also Zimmermann, Arch. der Phys. Heilk. 1848.

Pneumonia may have the same typical course as is observed in that of idiopathic origin.[1] That the intensity of these blood alterations, and particularly the increase of fibrine, should be so especially marked in Pneumonia, and may in part be referable to the peculiar relations of the organ (to which reference has been already made), is very probable. Pyrexia *per se*, independently of local inflammations, has not, except in the case of acute rheumatism, any marked proclivity to the production of hyperinosis. The large excretion of urea during the height of the pyrexia and its diminution during the progress of resolution (even where effete materials from the lung must continue to be absorbed into the blood), conclusively show that this phenomenon is due to increased tissue-changes throughout the system, produced probably by perverted nervous action, and which are only secondarily referable to the process in the lung. The increased destruction of red blood-corpuscles shown by the simultaneous increase of pigment in the urine, is perhaps referable to both the general and local conditions, since it frequently persists after the excess of urea has ceased to be observed. Zimmermann has further remarked that the decrease in their number, noticed by Andral and Gavarret, may be due not only to this cause, but to a subsequent defective formation arising from the abnormal conditions under which the white corpuscles are formed during the process of local inflammation and in the pyrexial period.

The disorder which on a lesser scale presents the greatest analogy with acute Pneumonia is perhaps acute tonsilitis, where we have the same short initial state, a similar intensity of rigor and prostration, a similar sudden invasion of pyrexia, and a similar rapid decline of this before the local inflammation has shown any signs of abatement. In tonsilitis also we have frequently an equal difficulty with pneumonia in verifying a distinct cause, and a certain amount of evidence at least exists in the case of the so-called "hospital sore throat," that it may also be produced by other poisons than those originating within the system from the impeded exercise of the functions of the skin.

The associated PATHOLOGY of Pneumonia has been already almost sufficiently described under the complications of the disease. A few points only deserve further attention.

In the Lungs themselves.—The mucous membrane of the bronchi is more or less injected, but the tubes seldom present much evidence of the dilatation observed in broncho-pneumonia. Plastic exudations, moulded to the shape of the tubes, are very common in the smaller bronchi. In some cases, however, this process may extend to the larger bronchi, which may be found thus obstructed through considerable areas.[2]

[1] See an interesting case from Mr. Hilton's practice, "Medical Times and Gazette," 1867, i. p. 144, where Pneumonia supervened after a broken rib. The temperature rose abruptly to 103°, and fell by crisis on the seventh day.

[2] This condition appears to have been first described by Reynaud, Mém. oblit. des Bronches, Arch. Gén. de Méd. 1835, iv. p. 157.

Acute Emphysema is sometimes observed in parts adjacent to the hepatized portion.

Œdema surrounding the consolidated part is more common, and may, by its extension and by its appearance on the opposite side, prove a source of much danger to life.

The Bronchial Glands are usually swollen and medullary in appearance. They are only in the worst instances subject to suppurative changes.

The Pleura is almost invariably inflamed when the hepatized part is situated at the surface of the lung. Effusion is, however, less common than the formation of false membranes.

In the Heart the complication of pericarditis has been already alluded to. In some cases this is due apparently to direct extension of the inflammation, for it is most common when a part of the left lung in juxtaposition with the pericardium is the seat of the Pneumonia. It appears, however, to arise sometimes under circumstances inexplicable by this cause, and it may then, according to the date of its appearance, be held to be due to the same cause as that in which the Pneumonia originated, or to the secondary blood-poisoning[1] caused by the absorption of the inflammatory products from the lung.

The right side of the heart is usually in fatal cases found distended and containing large and firm clots. Bouillaud[2] thought that Pneumonia predisposes to ante-mortem polypoid concretions, and this opinion is confirmed by Hasse,[3] who adds that he has found secondary infarcta in the spleen from this cause.

One of the most important consequences of Pneumonia on the circulation is the occasional occurrence of thrombosis in the pulmonary vessels leading to the affected part. This event, caused in all probability by the retarded circulation in the lung,[4] is not uncommon, and may, by extending to the larger branches of the pulmonary artery, be a source both of immediate danger from sudden death, and may also, in great probability, retard the process of resolution and the subsequent convalescence.

Catarrh of the Gastro-intestinal Mucous Membrane is by no means uncommon. The characters of the appearances found have been already described in the section devoted to diseases of the stomach. In some instances, however, this proceeds to a more serious stage by

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[1] Parkes, loc. cit.

[2] Traité Clin. des Maladies du Cœur, ii. 716.

[3] Loc. cit. 214.

[4] Virchow, Ges. Abhand. 222. It appears first to have been described by Baron, Arch. Gén. 1838, ii. 17, who first had the merit of distinguishing this event from the effects of inflammation of the coats of the artery. Malherbe, Journ. de Nantes, 1843 (Canstatt's Jahresb. 1843) first referred it to the retarded circulation. See also Mr. Paget's Memoir on this subject, Med.-Chir. Trans. xxvii. ; Carswell, Illust. Princ. Forms of Dis., art. " Mortification ; " Cruveilhier, Path. Anat., liv. xxxii. p. 2, who distinguishes the site of the coagulation as being in the artery, and not in the veins.

producing dysenteric ulcerations of the colon.[1] Hæmorrhage from the large intestines and stomach have been described by Barthez and Rilliet.[2]

The Liver is found congested, and the gall-bladder occasionally distended, but, even when icterus has been present, there may be no demonstrable obstruction of the ducts.

The Spleen is commonly congested, softened, pulpy, and opaque; characters which it presents after death in most of the acute febrile diseases.

The Brain rarely shows any other change than congestion; but in a few instances, when delirium has been violent, there has been found purulent infiltration of the subarachnoid space on the convexity of the hemispheres, and also of the base, which may also extend to the membranes of the cord. In many cases of delirium, however, the brain is found perfectly healthy.[3]

The influence of primary Pneumonia in the production of *other diseases* appears to be but slight.

That any permanent effect is produced on the heart appears to be disproved by Grisolle's statistics. Nor does the occurrence of Pneumonia, in the course of a cardiac disease already existing, appear to have any specially unfavourable effect upon the cardiac state. Its effect on *tubercular patients* appears, however, to be more doubtful. It is perfectly true, as Dr. Walshe has stated, that patients with tubercles already formed in the lungs may recover rapidly and completely from intercurrent acute Pneumonia, and Grisolle found that twenty-two patients of tubercular diathesis affected with Pneumonia all recovered perfectly.[4] In some cases, however, of tuberculosis, the convalescence is protracted and the cure imperfect, and in others the inflammation of the lungs tends to be followed by rapid softening and cheesy change. In fact, intercurrent Pneumonia must always be regarded as one of the greatest dangers of tubercular patients. Reso-

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[1] The result of Dr. Bristowe's observations on this subject (Path Soc. Trans. viii. 66) have led him to regard dysentery as a very common complication of Pneumonia. Out of 16 cases of acute primary Pneumonia proving fatal, he found dysenteric ulceration of the large intestine in four. The possibility of some epidemic influence may perhaps be regarded as not improbable in these cases, since the period over which part of Dr. Bristowe's observations extended included one of the recent epidemics of cholera.

[2] Loc. cit. i. 352.

[3] Grisolle. Louis, Fièvre Typh. i. 359, ii. 37. Immermann and Heller found that out of 30 cases observed in Erlangen during the years 1866 to 1868, nine presented post-mortem signs of meningitis. They attribute this condition in part to the simultaneous occurrence of epidemic cerebro-spinal meningitis; Deutsch. Arch. für klin. Med. v. (Virchow's Jahresb. 1868). Weber (Path. Anat. der Neugeborenen und Säuglinge, ii. 61) has also found cerebro-spinal arachnitis during an epidemic of Pneumonia.

[4] Huss also states, p. 24, that in northern climates acute Pneumonia has very little influence in the production of tubercle. He quotes, however, p. 162, from Gellersted "Bidrag till den Tuberculose Lungostens Nosographie och Pathologie," a statement that of 310 cases of phthisis, 23·5 per cent. had within a longer or shorter period suffered from one or more attacks of Pneumonia.

lution is imperfect—the affected parts tend to pass into grey consolida-tion, and in such parts fresh formations [1] of tubercles rapidly form and disintegrate.

DIAGNOSIS.—The diagnosis of the existence of acute Pneumonia essentially depends on the recognition of an acute febrile disease associated with the physical signs of consolidation of a portion of the lungs. Without this combination its presence cannot be affirmed with certainty in the earlier stages, though it must be remembered that patients may first come under observation at later periods, pre-senting the physical signs of consolidation of the lung, but after the initial fever has subsided. It is, however, important that its early stages should be recognised before the signs of consolidation are dis-tinct. Under this head certain phenomena connected with the mode of invasion deserve special prominence.

Among these perhaps the most important and constant is the pyrexia, which, although not pathognomic, still presents very marked and distinctive features, and is so invariable a symptom that the diagnosis of Pneumonia during the acute stage can scarcely be made in its absence. Whether or not the invasion be preceded by rigors, the sudden rise of temperature in a subject, previously non-febrile, should always excite suspicion, and it may be remarked that this rise of temperature may precede by hours, or even days, the appearance of the distinctive physical signs in the lungs. The use of the thermo-meter is also often a mode of recognising the invasion of Pneumonia when its symptoms are obscure, and appearing in the form of vomiting or convulsions in children, or of the prostration with which it often commences in old people. The rise of temperature in most of the acute febrile diseases is commonly gradual; in Pneumonia it is sudden, and maintains a higher elevation, during the first forty-eight or seventy-two hours, than is commonly seen either in these or in tubercular meningitis.[2]

The other phenomena of invasion which are most distinctive are the acceleration of respiration and the perversion of its ratio to the pulse. If to these and to the pyrexia are joined cough, rusty sputa, and pain in the side, the diagnosis of Pneumonia becomes one of infinite probability. Of the last-named symptoms, the relative acceleration of respiration is perhaps the most valuable, if, as Dr. Walshe remarks, hysteria be excluded, since expectoration may be absent, both in adults and children, or in the former the blood-stained tint may be wanting, and on the other hand, appearances of a very similar character to those seen in the first stage of Pneumonia may

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[1] These changes belong, however, more particularly to the history of phthisis; and their pathology, being in many points disputed, would involve too wide a discussion to be entered upon here, since by some authors the process of Pneumonia complicating phthisis is placed in a separate category of "catarrhal Pneumonia," or "infiltrated tuberculosis."

[2] Children are, however, liable to such sudden elevations of temperature from very slight causes, so that less reliance can be placed on this sign in them than in adults.

sometimes be observed in the sputa accompanying cardiac disease, and also in the early stages of congestive bronchitis. It may be noted, however, that in the last-named diseases fever may be entirely absent, or if present in bronchitis, the elevation of the temperature is rarely so considerable or so sudden as in the commencement of Pneumonia.

The distinctive features presented by the physical signs have been already fully described. When, however, in the commencement of the disease the inflammation first attacks the central parts of the lung, the signs of consolidation may be masked by healthy pulmonary tissue nearer the surface. Under these circumstances harsh breathing or weakened respiration may be the only phenomena observed.

Crepitation, when present, and when the possibility of œdema and of pulmonary apoplexy are excluded, is a valuable aid; but it is not unfrequently absent, and consolidation may take place so rapidly that it may not be heard in the earlier stages. As a rule it only furnishes further grounds for suspicion, until dulness on percussion, bronchial or tubular breathing, and bronchophony are established. The chief fallacy attending percussion is the occasional production of a quasi-tympanitic note over portions of lung, below which deeper-seated consolidation exists.[1] A comparison of the two sides is however, in children, often indispensable. Percussion of the chest of children should also be gently practised for reasons which I have already stated.

The superaddition of the auscultatory phenomena of the breathing and voice, and the increase of vocal fremitus over the affected part, if occurring collectively, render the diagnosis absolute; and as this combination of phenomena is the most frequent, Pneumonia may commonly be recognised with facility.

In exceptional cases, however, variations in these signs occur, which require some care in the diagnosis, particularly when one or more of them are wanting. This is sometimes the case in central Pneumonia, when the respiration may remain harsh or blowing, and crepitation and bronchophony may be absent. In some of these cases the diagnosis of Pneumonia can only be of relative value, depending on the presence of the characteristic pyrexia accompanied by rusty sputa.

The diseases of the lungs with which Pneumonia is most likely to be confounded are pleuritic effusion, œdema of the lungs, collapse, and certain forms of acute phthisis.

The question of the diagnosis of Pneumonia from *pleurisy with effusion* only occurs when the former affects the base of the lung or the whole organ.

In typical instances of the two diseases, the distinctive physical signs may be briefly contrasted as follows:—In *Pneumonia* the affected side is not distinctly bulged, and the intercostal spaces are not obliterated. Neither displacement of the heart, nor liver, nor

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[1] The tubular note over the larger bronchi may, however, sometimes prove deceptive to beginners.

diaphragm are observed. The dulness does not encroach upon the opposite side, or only to a very moderate degree. The dulness is less absolute than in pleuritic effusion, and has often a tubular tone. It does not change its site with the position of the patient, and the percussion note over the upper non-affected parts, though sometimes tympanitic, is never tubular or amphoric. The respiration over the affected parts is marked by a bronchial, or tubular, or metallic quality. The vocal resonance is strongly bronchophonic, and the vocal fremitus is increased. Crepitation may be heard in forced breathing or coughing.

In *pleuritic effusion* the side is bulged and increased in diameter, the intercostal spaces are obliterated, and may even be prominent, and fluctuation may sometimes be perceived over them. Displacement of the heart or liver, according to the side affected, is proportioned to the extent of the effusion. When this is extensive the dulness also encroaches on the opposite side. The percussion note is toneless, the sense of resistance is great. A tubular note, as observed by Dr. Walshe, is sometimes producible under the clavicle. In some instances the level and seat of dulness change with the position of the patient. The respiration below the level of dulness is weakened or absolutely suppressed. Weak, bronchial, or blowing breathing is heard near the spine and over the compressed lung. Vocal fremitus is diminished or abolished. Vocal resonance is also abolished below the level of dulness, and it is bronchophonic or ægophonic towards its limits. Friction may or may not be present.

Difficulties may, however, occasionally arise from exceptional combinations of the phenomena presented by each of these diseases. In the rare instances when, in Pneumonia, there are found, together with dulness on percussion, a simple absence both of the respiratory murmur and of bronchophony and fremitus, the most accomplished observers have been led into the error of mistaking the condition for one of pleurisy with effusion.[1] The signs which best distinguish Pneumonia under such circumstances, are the absence of the enlargement of the side, of the obliteration of the intercostal spaces, and of the displacement of the various viscera, which characterise extensive effusion. Variation of the percussion dulness with the position of the patient, may, if observable, serve as a further aid if pleurisy be present, though its absence cannot always be relied upon for the exclusion of this affection. As a sign of minor value, it may be stated that the percussion note is more absolutely toneless in pleurisy, and seldom, if ever, has the higher pitch of that observed in Pneumonia. The tubular or amphoric note over the upper part of the lung sometimes heard in pleurisy is not, as observed by Dr. Walshe, met with in the non-affected upper portions of the lung when Pneumonia exists at the base; the percussion here, though hyper-resonant, being commonly of a lower pitch, and sometimes tympanitic in quality. The invasion of the pyrexia is commonly less acute, and the tempera-

[1] See Barthez and Rilliet, i. 589 ; also Wintrich, before quoted.

ture less elevated in uncomplicated pleurisy.[1] It is, however, an event of the extremest rarity that the absence of respiration and the diminution of the vocal fremitus and resonance are, as a matter of practice, found to co-exist simultaneously over a pneumonic lung. In doubtful cases, the fremitus may aid in distinguishing the two diseases, being increased in most cases of Pneumonia and diminished in pleuritic effusion. In children, and when in adults and females the voice is weak, this sign may be comparatively indistinct.

In the cases of pleuritic effusion, where bronchial breathing and bronchophony persist, the diagnosis from Pneumonia may also commonly be made by the signs above enumerated. The fremitus may be a further guide,[2] and, as Dr. Walshe has remarked, the true tubular respiration of Pneumonia is wanting in pleurisy, and the indistinct bronchial breathing heard is most commonly met with near the spine.

Œdema of the lungs, which may be attended by the crepitant râle of pneumonia, may commonly be distinguished from it by the absence of pyrexia, by the minor degree of dulness, by the respiration being simply weak, and by the concomitant affections in which it originates.

The diagnosis of simple Pneumonia from *acute phthisis* when the latter is only attended by the disseminated formation of miliary tubercles, is comparatively easy, owing to the absence of dulness in percussion in the latter affection. When, however, acute tuberculosis is attended by, or commences with, a rapid and extensive pneumonic infiltration, the diagnosis may be almost impossible during the early stages of the affection. This, however, is less common in acute phthisis than a more gradual extension of the pneumonic process, which usually takes place irregularly and through longer periods than are observed in primary Pneumonia. The pyrexia of acute phthisis is more irregular in its course; it has more marked remissions than those of Pneumonia, and the exacerbations often occur at very varying periods of the day, the maximum temperature being attained on one day in the morning and on another in the evening,—a phenomenon of the extremest rarity in primary Pneumonia. Much depends on the time at which the case comes under observation. If at a later period than the first ten days, the protraction of the pyrexia may always be regarded as a suspicious circumstance. If the fever is very irregular in its course, and acute exacerbations with very marked remissions occur at uncertain intervals, the suspicion is still further strengthened, since in most cases, when the pyrexia of a simple Pneumonia is retarded in its final disappearance, the fever maintains as a

[1] This sign is of minor value in relation to cases of Pneumonia of moderate severity, and when the temperature does not rise above 102°. It should also be remembered that the Pneumonia may come under observation for the first time after the fever has subsided. Under these circumstances the diagnosis from pleuritic effusion may depend on the physical signs alone.

[2] Dr. Walshe, however, states that fremitus may be diminished in extensive hepatization, though not to the same extent as over an equal amount of effusion. He adds that he has often known fremitus feeble, and vocal resonance strong over effusion, but that he has never met with this combination in hepatization. The value of these signs in diagnosis depends on their combination, and but little reliance can be placed on either singly.

whole a low standard, and subsequent elevations of temperature to 103° or 104° are very rare. Pneumonia of the apex, running such a course, is still more open to suspicion than that affecting the base of the lung.

If, in addition to these symptoms, signs of the formation of cavities become increasingly apparent, the grounds for an unfavourable opinion are still further strengthened, though doubt may still exist, owing to the possibility of the formation of abscesses in the hepatized tissue. Evidences of progressive disease in other parts of the lung at a late period of the case are still more serious symptoms; and the implication of the opposite side, particularly if general râles appear here accompanied by irregular spots of consolidation, and by signs of destruction of tissue, will, together with the conditions of pyrexia before alluded to, and in conjunction with rapid emaciation and other signs of hectic, render the diagnosis of tubercle almost a certainty. Louis believed that implication of the anterior and superior parts of the lung, without the invasion of the whole apex, was almost certainly an evidence of tuberculosis, but this statement, though affirmed by Barth and Roger, is disputed by Grisolle.[1]

The diagnosis from *Collapse of the lung* will be considered under the head of Broncho-pneumonia.

There are certain other diseases with which acute Pneumonia is occasionally confounded.

The sudden prostration, with severe headache and high degree of pyrexia at the outset, not unfrequently simulate *Typhus;* so much so that from the reports of the different fever hospitals it would appear that a certain number of cases of Pneumonia are annually sent to these institutions under this error. Even in the earlier periods the mistake both from typhus and typhoid may be avoided, as Dr. Grimshaw has remarked, by the observation of the temperature, which rises suddenly in Pneumonia, but in the continued fevers rarely attains its maximum before the sixth or seventh day. At the later periods the physical signs of consolidation of the lung on the one hand, and on the other the appearance of the characteristic rash of the continued fevers, are sufficient to prevent mistakes. The existence of herpes may also serve as a clue to the nature of the affection, being very common in Pneumonia, while it is scarcely ever met with in the course of the continued fevers.

Pneumonia commencing with cerebral symptoms in children may be easily overlooked, particularly when it affects the apex of the lung.

Ziemssen has remarked that *tubercular meningitis* rarely gives at the outset the high temperature of acute Pneumonia. The remissions are also more marked. They are more variable in their extent, sometimes showing a range of temperature of 1·8°, 2°, or even 3° Fahr., and

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[1] Dr. Walshe also affirms that Pneumonia having this position is commonly, but not always, tuberculous (loc. cit. p. 497).

[2] See Dr. Grimshaw, Thermometric Observations on Pneumonia; Dublin Quart. Journ., May 1869.

the pyrexia is less continuously maintained. Some differences also in the character of the nervous symptoms have been already alluded to.

The diagnosis of the *different forms of consolidation* rests upon no absolutely reliable signs.

The stages of *grey hepatization* and of *suppuration of the lung* cannot be determined absolutely by the duration of the disease.

The prune-juice diffluent sputa, which were thought at one time to be characteristic of the former, have been shown to be by no means pathognomonic of this state, though their appearance affords strong ground for suspecting its presence.

It may, however, be strongly suspected when the amount of sputa is much increased, and when, instead of being rusty and tenacious, they become profuse, diffluent, and puriform, and still more so when they are fœtid and offensive. Protraction of the period of resolution, attended by coarse metallic râles in the chest, and by extreme prostration, pyrexia and delirium, afford, together with the signs derived from the sputa, additional evidence of this condition of the lung.

The diagnosis of *abscess* can only be made when the expectoration of puriform matter is sudden and copious. The detection of elastic fibres in the sputa affords a further proof of its existence. *Gangrene* can only be suspected when great prostration, together with extreme fœtidity of the sputa, occur late in the disease; the only positive proof of its existence depends on the discovery of débris of the pulmonary tissue in the sputa, but in these, elastic fibres are seldom distinct.

THE PROGNOSIS of Pneumonia in relation to its general mortality has proved to be the same insoluble problem that Andral[1] pronounced it, when he drew attention to the fact that the death rate in different statistics varied from 33 to 2 per cent. The difficulty has, however, still further increased in later years by the varying and contradictory statistics of the result of the different methods of treatment adopted for the disease. The results attained by Dr. Bennett,[2] who in 129 cases, of which twenty-four were complicated, had the good fortune to see all recover except four which presented serious complications, are so singularly favourable that they might lead us to regard the disease as less dangerous than it sometimes proves to be. Even in young male subjects of previously good health, Pneumonia may, as I have seen, sometimes falsify the hopes entertained from the relatively small mortality of such cases; and my own hospital experience has yielded a much greater proportion of fatal results than Dr. Bennett's, though the general methods of treatment have been very similar to his. In fifty-five cases which I have observed or collected from the case-books of University College Hospital, and the North Staffordshire Infirmary,[3]

[1] Cours. de Path. Méd. 1836, i. 386.
[2] The Restorative Treatment of Pneumonia, 1866. A very similar result is recorded by Dr. Waters, Dis. of Lungs, p. 87, who in forty-four uncomplicated cases only met with one death.
[3] I do not present these as statistics of these hospitals, as I cannot feel sure that they embrace all the cases admitted.

I have met with eight deaths, but in all these the attendant circumstances of the disease were such as fully to account for the mortality.

One was in an infant of five months, in whom the whole of one lung had passed by the fifth day into a state of grey hepatization.

One was in a young female, where the Pneumonia was apparently developed under the influence of some intense blood-poisoning, being complicated with parotitis occurring on the ninth day, and where there were also albuminuria, pericarditis, and constant vomiting, dysenteric diarrhœa, and a petechial eruption under the skin, which latter in some places passed into large vesicles filled with a dirty-looking blood-stained serum, and where also disseminated spots of a gangrenous character were found in both lungs.

In two other cases there were old-standing renal disease and recent pericarditis. In one, an old woman, the bronchi were calcified, and there was extensive pleuritic effusion on the opposite side and thrombosis in the pulmonary artery.

Two others also presented extensive double Pneumonia: in one, a man aged 60, there was also an adherent pericardium and a fatty heart. The other, a young man, had been a hard drinker, and was suffering from syphilis.

In the remaining case there was also general bronchitis with emphysema, and the whole of one lung was in a state of grey hepatization.

Pneumonia, when extensive, certainly carries with it conditions which may prove fatal whatever the treatment adopted. It may kill by the intensity of the cause in which it originates, or by the secondary lesions to which this may give rise, particularly in the pericardium and in the kidneys. It may prove fatal by asphyxia, especially when the affection is double, or is complicated by old-standing emphysema, by extensive general bronchitis, by œdema of the lung, or by pleuritic effusion of the opposite side ; and finally, and particularly in elderly people of weak constitution, death may take place in spite of the most energetic restorative measures, and when no previous lowering treatment has been adopted, in the prostration following the crisis, which may pass into fatal collapse.

It must be remembered, also, as stated under the etiology of the disease, that the mortality varies greatly in different years under the same methods of treatment. This is seen markedly in Huss's statistics, where, under an "antiphlogistic" treatment, the relative numbers of 9·1 and 14·1 per cent. may be observed; and after this plan had been abandoned the mortality in different years may yet appear as 6·1 and 13·4 per cent. The returns from the Julius Hospital of Würzburg[1] for the triennial periods of 1854-7 and 1857-60 show a similar difference; the mortality in the former period being 11·2 per cent., and in

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[1] Bamberger, Wien Med. Woch. 1857, No. 5; Roth, Würzb. Med. Zeit. i. Nos. 3 and 4.

the latter 18·9 per cent., the conditions of treatment in both periods being very similar. Brandes,[1] in Copenhagen, found the mortality in two successive years vary to the degrees of 5·4 and 31·0 per cent. The same fact is borne out by the returns of the Registrar-General before alluded to.

The most important etiological conditions which influence the mortality of Pneumonia are the age of the patients, their previous health and habits of life, their sex, the extent of the disease, and, to a less extent, its seat and the existence of complications.

Age.—It was formerly thought that the Pneumonia of infancy and childhood was an excessively fatal disease,[2] but these statements rested probably in the first place on the confusion between Pneumonia and collapse of the lung, and in the second in no small measure on the severe antiphlogistic treatment then adopted. Strangely in contrast with this belief is the remark by Barthez and Rilliet, that the opportunities for post-mortem examination in the acute lobar Pneumonia of children are excessively rare.[3] The statistics of Ziemssen and Steffen bear out these assertions. The former, out of 201 cases of Pneumonia in children, only lost seven in the acute stage. In four others the recovery was imperfect, and two of these died, giving a total mortality of less than 4½ per cent. Steffen, in 94 cases, lost 13.[4] It would appear from his tables that the mortality is greatest in early childhood, since nine of these were under three years of age.

The period of dentition, though showing from the results of Steffen a greater mortality than the later years of childhood, does not in Ziemssen's opinion unfavourably influence the prognosis, if *all lowering treatment be withheld.* This state tends, however, to be accompanied by a higher degree of pyrexia and by more severe cerebral symptoms.

After the period of childhood the mortality remains comparatively low until the age of 30 is attained, but after this it rapidly advances

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[1] Virchow's Archiv, xv. 213. Brandes very properly solves part of this enormous difference by the explanation that the higher mortality was due in the latter instances to the patients with delirium tremens admitted under his care. The number of cases entered in the two years were respectively 55 and 87, and out of the 27 fatal cases in the latter period, 12 were instances of delirium tremens complicated with Pneumonia; five others were cases of typhoid fever with Pneumonia, and in five more, complications with "organic cardiac disease" were present.

[2] Thus Valleix (Mal. des Enf. nouveaux-nés, pp. 45, 47, 70) says, that of 128 cases collected by Vernois and himself, nearly all died.

[3] Mal. des Enfants, i. 515. Barthez and Rilliett (ib. p. 535) say that in hospital they lost one-seventh, and in town practice one-eighth of their patients, but this remark appears to apply to primary and secondary pneumonias collectively. The previous health and earlier treatment of the patients in private practice would appear to be sufficient to explain the difference. They distinctly refer the deaths of some of their patients to "poisoning" (*sic*) by tartar emetic. Barthez (Bull. Akad. Med. 1862, vol. xxvii. p. 676) gives a further report on this subject, stating that among 212 children aged from 2 to 15, the subjects of Pneumonia, only two deaths occurred.

[4] Some of Steffen's cases were secondary to measles, scarlatina, and variola. Others were complicated with other diseases. Of his uncomplicated cases, 88 in number, he only lost 7.

with each succeeding decade;[1] so that Marc D'Espini's statement may be regarded as approximatively true, that in more than half the patients dying of Pneumonia the age exceeds 50;[2] while Prus showed that in 129 cases whose age exceeded 60, 77—or 59 per cent.—died,[3] and Hourmann and Dechambre[4] give a nearly equal proportion.

Sex.—Pneumonia is a more fatal disease to females than to males. Huss gives the relative mortality as, males 10 per cent., females 14 per cent. The returns from Vienna show the mortality to be as 2 to 3 in the male and female sexes, so that although Pneumonia is a less common disease in the female sex it is proportionately considerably more dangerous. The disease also in the female sex appears to be more protracted, showing an average duration of three days in excess of that observed in the male, in the cases which recovered. Females are also, according to Huss, more liable to double Pneumonia than males. His tables also appear to show that the mortality in the female sex is less influenced by age than in the male.

Certain conditions peculiar to the female sex appear to add to the dangerous characters of Pneumonia in them, though these are scarcely sufficient to explain the whole of the relative difference.

The condition of pregnancy appears to render Pneumonia peculiarly dangerous. Eight out of 18 cases collected by Grisolle proved fatal, and this author remarks that abortion is more liable to occur in its course than in that of any other acute disease, with the exceptions of variola and cholera. Pneumonia occurring in the puerperal state has also an extreme gravity. Menstruation, according to Grisolle, increases the intensity of Pneumonia occurring during this period, though without necessarily adding to its mortality. The state of chlorosis, according to Huss, appears in some degree to afford a protection against Pneumonia, but imparts to it when present an element of additional danger.[5]

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[1] Among the mass of statistical evidence on this subject, the following table from Huss (p. 93) gives probably the most reliable data :—

Age.	No. of Cases.	No. of Deaths.	Percentage.
5—10	9	1	11·11
10—20	229	14	6·11
20—30	1,041	61	5·85
30—40	816	97	11·88
40—50	363	72	19·83
50–60	127	27	21·60
60—70	29	7	24·13
70—80	4	2	50

[2] Ann. d'Hygiène et Méd. Leg. 1840, xxiii. p. 50.
[3] Mém. Acad. Méd. 1840, viii. 13.
[4] Arch. Gén. xii. 28.
[5] Of twenty-five cases of this complication met with by Huss, five, or 20 per cent., died.

The extent of lung affected increases, *cæteris paribus*, the gravity of the affection in a manner which may be readily understood, though a limited area of inflammation may, when unfavourable complications exist, prove equally dangerous. Double Pneumonia must, *à fortiori*, be always regarded as a source of very serious danger from the extreme impediment to the respiration involved by it, the mortality from this condition being by universal consent regarded as double that of the unilateral disease.[1]

Pneumonia of the apex was, especially by the authors of twenty years ago, regarded with peculiar distrust.[2] Grisolle states that the mortality in patients so affected, and under 40 years of age, is, when compared with that of the base, as 5 to 3. Louis[3] regarded it as one of the elements of the increased mortality in the aged. Barthez and Rilliet speak of it in children as being especially liable to be associated with dangerous cerebral symptoms. Ziemssen[4] also, and Gerhardt, although recognising the comparative frequency of nervous disturbance attending Pneumonia of this site in children, do not regard it as being ultimately of unfavourable augury. Some doubt, however, still exists regarding its specially unfavourable character in adults.

The occurrence of grey hepatization is of very unfavourable significance. Huss states that one-third of the patients perished in whom its presence could be probably presumed. It usually, at least when occurring early, signifies a more rapid progress of the disease and a weaker resisting power of the individual. In the later stages it implies defect in the restorative powers which conduce to resolution.

Gangrene in the course of acute Pneumonia is of very serious augury. Of twelve cases occurring in Huss's practice only two recovered.

Sestier[5] and Briquet[6] both thought that Pneumonia was more dangerous in *cold seasons*. Grisolle disputes the validity of these data, and points out that in Briquet's cases a large proportion of the mortality was due to the advanced age of the patients, and concludes that season has but little influence in any other respect. Huss's statistics, however, show the remarkable fact that though Pneumonia is less frequent in the last half of the year, yet that the mortality during this period is by far the greatest, in the proportion of 17·6 per cent. for the later six months to 12 per cent. in the earlier, while the excess during the last half prevails during each individual month. The contrast is still greater for some months : April, which yielded 355

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[1] See Grisolle, loc. cit. Huss (loc. cit.) gives as the collective mortality—double Pneumonia, 22 per cent.; right unilateral Pneumonia, 9 per cent.; left ditto, 7·9 per cent. Huss's tables show further, in respect to age, that while double Pneumonia is most common from 20 to 30, the mortality from it is greatest from 40 to 70. The mortality from double Pneumonia appears to be about equal for both sexes.

[2] Chomel, Dict. de Méd. xxv. 158. In 55 deaths he found 13 of the upper lobe, 11 of the lower, and 31 of an entire lung.

[3] Rech. sur les Effets de la Saignée, 42.   [4] Loc. cit., 211.

[5] Chomel, Lec. Clin. Méd., Pneumonie, p. 445.

[6] Arch. Gén. de Méd. 3ᵉ Sér., 1840.

cases, showing only a mortality of 8·7 per cent.; while August, with only 113 cases, had a death-rate of 25·6 per cent. The cases occurring during the hot months also presented greater severity, a condition considered by Huss to be partly due to the liability during these to gastro-enteric catarrh, and also to a larger consumption of alcoholic fluids at this season.[1]

Previous attacks do not *per se* increase the danger of the disease. The more advanced ages at which later attacks may occur in adults, do, however, somewhat increase their risk.

It was at one time thought that Pneumonia was most dangerous in robust individuals; but Huss's statistics have most clearly disproved this, and show that the most dangerous forms of the affection, both clinically and pathologically, occur in patients of weakened constitutions.

Of all conditions, however, which, independently of other circumstances, impart a special danger to the disease, *habitual drunkenness* must be regarded as one of the most serious. The mortality from Pneumonia under these conditions is nearly double that ordinarily observed, amounting to 20 or 25 per cent.[2]

The existence of *complications* forms the most serious element in the prognosis, and most largely influences the mortality of the disease. This is sufficiently apparent from Huss's collected results, where the mortality of the non-complicated cases was only 5·79 per cent., while that of the complicated cases amounted to 19·29 per cent. The relative danger of the various complications, as observed by him, will be best seen in the table before quoted (see p. 657). It is, therefore, only necessary here to remark that of the most ordinary of these, the greatest mortality occurs in the presence of endocarditis (75 per cent.); pericarditis (54·5 per cent.); Bright's disease (50 per cent.); old valvular disease of the heart (30 per cent.); tubercle (33·3 per cent.); emphysema of the lung (23 per cent.); chlorosis (20 per cent.); and chronic alcoholism and drunkenness (25 and 20 per cent.). The danger is proportionately increased if more than one complication occur in the same patient. It may further be noticed that certain complications, such as rheumatism and erysipelas of the face, do not appear unfavourably to influence the general course of the disease, while both bronchitis and pleurisy do so to a less degree than might be *à priori* believed.

An extreme degree of pyrexia is considered by many an unfavourable sign. Wunderlich regards a temperature of 104° Fahr. as the limit of mild cases. It must, however, be remembered that cases may prove fatal in whom this temperature is never attained.[3] Wunderlich regards a gradual rise taking place after the fourth day as

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[1] The returns from the General Hospital of Vienna show that in some years the mortality is greatest in the winter months.

[2] Huss, loc. cit.

[3] This is further confirmed by Griesinger. Of thirty fatal cases the temperature only reached or exceeded 104° in eight. A temperature of 105·2, occurring in only one instance, was the maximum attained among these fatal cases. (Bleuler, loc. cit. p. 33.)

a very unfavourable symptom.[1] The rapidity of the breathing has less influence on the prognosis than that of the pulse, but a very rapid respiration associated with a low temperature is pointed out by Wunderlich as indicative of danger. Irregular respiratory movements show a severe implication of the nervous system. Excessive dyspnœa amounting to orthopnœa, particularly when associated with cyanosis, are also indications of considerable gravity.

A pulse above 120 always indicates weakened cardiac powers, but its unfavourable augury is less in young children than in adults. In the latter, a pulse above 130 or 140 is a sign of great danger, and particularly when the temperature is not markedly high.[2] Grisolle says that all his cases died in whom the pulse exceeded 150. Extreme dichrotism of the pulse has a very similar import. It has been already stated that in some cases the pulse may be markedly retarded before a fatal issue. Irregularity and intermittence of the pulse except in elderly people, in whom these symptoms are not uncommon, must also be regarded as suspicious symptoms.

Few signs can be drawn from the sputa. Those of liquorice or prune juice tint are the more serious, but do not necessarily indicate a fatal issue. The serious import of profuse hæmoptysis has been before alluded to. Diffluent puriform expectoration when profuse in the later stages of the disease, and when associated with great prostration and persistence of the physical signs, are also symptoms of considerable gravity. Suppression of the expectoration from weakness, together with increase of tracheal râles, is of very serious augury. The total absence of expectoration throughout the disease has no influence on the prognosis.

Marked disturbances of the nervous system are always indicative of the severity of the disease. A mild degree of delirium is not uncommon in children, and also in elderly people; but in the latter it is a serious symptom.[3] In adults, however, severe delirium is always dangerous, particularly when occurring late in the disease, or when habits of drinking have preceded the attack. Convulsions, repeated after the onset of the disease, are in children a sign of much danger.

Intense prostration with sunken and pallid features, and profuse sweating, are always suspicious, and have a gravity proportioned to their degree. In the more marked forms of so-called Typhoid Pneumonia, the prognosis must always be doubtful.

Total suppression of the chlorides in the urine indicates a greater severity of the disease than when these are present, but does not, independently of other circumstances, materially affect the prognosis. Severe gastric disturbance and diarrhœa have a very similar import.

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[1] Die Eigenwärme in Krankheiten.

[2] Bleuler, loc. cit. Of adults with a pulse above 120, one-third died whose age did not exceed 40; over ætat. 40 one-half died; over 60 all died.

[3] Bleuler (loc. cit.) observed a mortality of one-fourth of patients under ætat. 40 who exhibited marked delirium; over 40, three-fourths of these died.

Their effect is certainly unfavourable, but it can only be judged of in relation to the general strength of the patient. Icterus does not necessarily increase the gravity of the prognosis.[1]

A protracted defervescence with a high pyrexia are also unfavourable. The liability to relapses in the early days succeeding the crisis should also impose caution against a premature confidence in the cessation of danger.

The terminations in a chronic state are so excessively rare that they hardly form an element in the consideration of ordinary forms of acute Pneumonia. The possibilities of a more protracted course are sufficiently shown in the previous account of the ordinary progress of the disease.

The occurrence of herpes appears from the researches of Griesinger[2] and Geisler[3] to have a favourable prognostic signification.

TREATMENT.—There is, perhaps, no subject in modern medicine which has been more earnestly discussed of late than the treatment of Pneumonia. It has been the *champ de bataille* between the advocates on the one side of "heroic" measures, and the supporters of a "rational" and of "expectant" treatment on the other; and since the first-named methods have been, to a large degree, shown to be worse than useless, the question has become further complicated by the assertion that a change of type has ensued, by which the nature and characters of inflammatory diseases in general have been, during recent years, materially modified.

When, however, the natural course and the various relations of this disease are attentively considered, it is apparent that no malady can well be chosen less suited to afford logical proof, by means of statistics, of the relative value and the curative effects of any system of treatment applied indiscriminately, though the reverse is more easily shown by the enormous excess of mortality which prevails when an "active" treatment is universally employed.

An acute disease with a natural tendency, under favourable circumstances, to terminate spontaneously by a sudden crisis occurring at periods varying from the 3rd (or even the 2d) to the 7th or 11th days, presents the most singular elements of fallacy in reasoning from the beneficial effects of active medical interference. If to these we add the manner in which its mortality is affected by age, by constitution, by sex, by the presence or absence of complications, and by the other but unknown epidemic conditions which have no slight effect in the same direction, it would appear a task of the extremest difficulty to collect sufficient data in order to institute a logical comparison between patients under similar circumstances of the disease, but under different systems of treatment, so as to form any true con-

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[1] This is the almost universally adopted opinion. Bleuler, however, records a mortality of 35 per cent. of cases in which icterus was observed.

[2] Arch. der Heilk. 1860, vol. i.

[3] Ibid. 1861, vol. ii.

clusion as to the relative value of the methods to be adopted for its cure.

Looking to the evidence of statistics, and to the individual experience of careful observers, it must be admitted that medicinal interference and active treatment are, collectively speaking, of but little influence, either in shortening the duration in, or diminishing the mortality of, Pneumonia. Treatment, in its wider sense of nursing, diet, support, and remedies adapted to individual cases, is however, the author believes, by no means inefficacious in aiding the tendency of nature to effect a cure.

The remedy which has been most extensively adopted, but which has been almost completely discarded of late in this country, is *venesection.*

Reintroduced by Sydenham[1] as applied both to pleurisy and pneumonia, with the statement "Hujus morbi curatio in repetita venæsectione fere tota est," and supported by Huxham and Cullen, the amount of blood taken by these authorities and their followers in the treatment of this and kindred disorders was enormous. Day by day, with the progress of the disease, fresh venesections were practised, and Dr. Gregory, after bleeding a young man into convulsions by the abstraction of between 4 and 5 lbs. of blood in three days, considered that he had thereby cured him of pleurisy.[2] Bouilland recommends a daily bleeding to the amount of 14 or 16 oz. until the disease is cured. Andral asserts that no period of the disease contra-indicates venesection provided the other symptoms appear to require it, and that age is no barrier to this treatment, citing in its favour at advanced ages the authority of Frank,[3] and that it is to be applied to children equally with adults : the slightest threatening of a relapse called in his opinion for further bleeding : it is not to be omitted without the greatest danger, even if menstruation be present : profuse sweating is no contra-indication, nor is any amount of prostration to prevent it, if the respiration be seriously impeded :[4] it is to be equally practised in the secondary pneumonias of measles, variola, and scarlatina, though with caution in typhoid fever : it is only contra-indicated in the adynamic forms of the disease, and in some rare cases of special idiosyncrasy, and in the absence of expectoration. Grisolle, even for more moderate bleedings, recommends the abstraction of from 2 to 4 lbs. by repeated venesections, and still regards this plan as the most successful in the treatment of the disease.

The treatment thus indicated continued in use with more or less freedom in this country until attention was forcibly drawn by Dr. Balfour[5] to the lesser mortality of Pneumonia in Skoda's practice, and

[1] Works, Syd. Soc. Ed. p. 352.   [2] Quoted by Dr. Alison.
[3] Grisolle similarly quotes Morgagni (Epist. xx.), who bled a man over 80 with "success."
[4] On this point he quotes Stott, who bled for the eighth time a patient covered with petechial eruption.
[5] Notes in the practice of Skoda, Edinburgh Medical and Surgical Journal, 1847, p. 397. Brit. and For. Med.-Chir. Rev. 1846, vol. xxii. p. 590.

also in some of the homœopathic hospitals where bleeding had been for some time discontinued. Even before this period Becquerel[1] had shown the inutility of venesections in the pneumonia of children, and it is stated, on the authority of Legendre,[2] that Biett and Magendie had pursued an expectant treatment in Pneumonia with excellent results. Dr. Graves[3] had also asserted that the large bleedings practised by some physicians were unnecessary, and that repeated venesections were injurious in the disease ; but the statistics of Skoda's practice showed for the first time the striking contrast between the " heroic " and the "expectant" methods ; for while the mortality in 384 cases treated by him with small doses of *extractum graminis* and nitre was only 13·7 per cent., that of the Edinburgh Infirmary during a nearly corresponding period of five years was 35·9 per cent. Dietl's[4] comparative statistics, which appeared almost simultaneously with Dr. Balfour's papers, seemed still more forcibly to bring into contrast these systems of treatment, and may be said to have at once exercised an important influence on medical opinion both in this country and in Germany.

The arguments against bleeding have subsequently been most vigorously supported by Dr. Todd and Dr. Bennett, who have at least the merit of showing that the treatment by venesection is in most cases unnecessary, and that in a very large proportion it is positively injurious, and the same conclusions have been more or less completely adopted by the majority of the physicians of the present day.

The conclusion which has been practically arrived at by the medical profession with respect to the influence of venesection in Pneumonia may be, with approximative truth, expressed in the following terms :[5]—

(1) That indiscriminate bleeding immensely increases the mortality of the disease.

(2) That it is specially fatal in old people and in young children, in patients of exhausted constitutions, and in those suffering from chronic diseases, and particularly from Bright's disease.

(3) That it is absolutely unnecessary in the majority of cases of young adults and also of young children.[6]

(4) That in the vast majority of cases it has no influence whatever either in cutting short the disease,[7] or in lessening its duration,

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[1] Sur l'Influence des Émissions sanguines et des Vésicatoires chez les Enfants, 1838.

[2] De l'Expectation dans la Pneumonie. A posthumous memoir : Arch. Gén. 1859 xiv. 283.

[3] Clinical Medicine, 1843, ii. 42.

[4] Der Aderlass in der Lungen-Entzündung.

[5] See Appendix F.

[6] This is especially seen in Ziemssen's treatment, and also in a memoir by Barthez, who, in 212 cases of young children with lobar Pneumonia, only met with two deaths. Barely one-sixth were subjected to active treatment. (Arch. Gén. 1859.)

[7] This is most strongly affirmed by Louis and Andral, and also illustrated by the case by Zimmermann before quoted. Chomel (Dict. de Méd. xxv.) held that it might sometimes effect this.

or diminishing the pyrexia, but that occasionally these results appear to follow from its use when practised early.

(5) That in the majority of cases it hinders the critical fall of temperature and delays convalescence.

(6) That in the majority of cases, as shown especially by Dr. Bennett's and Dietl's data, recovery is equally, if not more rapid, when it is not practised as when it is resorted to.

(7) That in a few cases a moderate venesection may be necessary in the early stages to avert immediate danger of death from asphyxia.

Individual cases where apparent success has followed venesection are really but little capable of proving its general utility. It is a treatment to which I have never but once resorted, and have very rarely seen practised, and I can certainly affirm that those cases which may occasionally offer the strongest temptation to the use of the lancet recover just as well when it is withheld. The mortality among the cases which I have mentioned as coming under my own observation, has certainly been in such as would not, with any modern knowledge, have been considered fit subjects for venesection. Even the relief of dyspnœa, which is thus effected, is proved by universal consent to be only temporary in its duration, for this symptom usually results more from attendant œdema of adjacent portions of the lung than from the actual obstruction to respiration in the part affected by the primary disease unless this be very extensive; and as the amount of fluid withdrawn by venesection is speedily replaced by the absorption of water, the pressure on the collateral circulation of the lung is thereby only very temporarily diminished. It was, however, to this cause that the repeated venesections of former times were probably due, a system whose impropriety it is scarcely needful to discuss further.

Its employment in severe pyrexia is also shown by Ziemssen to be unnecessary, for though he admits that he has occasionally resorted to its use when the temperature has appeared dangerously high, he yet states that other cases of a similar nature recover equally well without it. I have already stated that the fatal cases which have come under my own observation have not in any instance presented this phenomenon.

When we consider, therefore, that the most urgent symptoms of the disease—the dyspnœa and the pyrexia[1]—are only temporarily diminished by venesection, and that they both tend in most cases to return after a few hours, the reasons for the adoption of this method of relief lose much of their validity.

It may be possible that cases of such extreme urgency may occasionally arise that venesection may be absolutely necessary to avert immediate death by apnœa. Such cases I must believe, however, judging from my own experience, to be excessively rare; though, in the event of their occurrence, this remedy is probably the best that

[1] See Appendix E.

could be adopted, and should not be shrunk from if the indications are urgent, but I believe that such a condition is the only one in which it can be regarded as absolutely necessary. The mortality from Pneumonia has appeared to me to depend much more on prostration in the later periods, than on asphyxia in the earlier stages of the disorder ; and the former result appears to be much more likely to occur when the strength of the patient is weakened by venesection. If, therefore, venesection appears to be positively required at an early period of the attack, the amount of blood withdrawn should be moderate, and should not exceed six or eight ounces.

With regard to the possible effect of this treatment in cutting short the disease, it may be stated that the chances in any given case are strongly against such a result. Looking at the general effects of this procedure, patients will, on the whole, be probably in a worse condition for passing through the later stages of disease when weakened by an artificial loss of blood than they are likely to be if their resources in this respect are husbanded : and though its dangers are the least in the case of young adults of good constitution who commonly "bear" bleeding comparatively well, this "tolerance" of the remedy by such subjects affords no proof of its general advantageous effects.

Most of the other methods of treatment directed immediately to the cure of Pneumonia afford nearly equal proofs of their inutility.

The comparative effect of large doses of *tartar emetic* is shown by Dietl's statistics,[1] while Rasori's[2] mortality from this method was 22 per cent., and Grisolle's 18 per cent.,—or in those treated exclusively by this method, 13 per cent. Independently also of this considerable mortality, the poisonous effects of the remedy were very frequently observed. Laennec spoke very highly of tartar emetic in more moderate doses, and considered that it had reduced the mortality from Pneumonia in his practice to a minimum ; but grave doubts have been thrown on the accuracy of Laennec's details[3] in respect to this method. Laennec asserted and Grisolle believes that it is more useful when preceded by bleeding. Louis[4] also and Trousseau[5] speak favourably of its results, but the data given by the former, complicated as his treatment was by venesection, afford but little proof of its efficacy.

Regarding the statements made, particularly by Grisolle, respecting its effects in Pneumonia, it cannot be denied that tartar emetic produces occasionally a feeling of relief to the patient, and in some cases lowers the frequency of the pulse, and apparently diminishes the pyrexia.[6] This effect, however, requires to be very carefully watched. It is a depressing agent both to the nervous system and to the cir-

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[1] See Appendix E.
[2] Ann. de Thérap. 1847, and in Archiv. Gén. 1824.
[3] See Grisolle. [4] Rech. sur la Saignée.
[5] Dictionnaire de Médecine, art. "Antimoine."
[6] Accurate thermometrical observations on this point are wanting.

culation, and is liable to increase the dangers of the later collapse. As far as my own experience goes, I believe that it is a remedy which can only very rarely prove of essential utility, and certainly, to say the least, the vast majority of patients will recover as well, if not better, without its use; and it is absolutely inadmissible in the adynamic forms of the disease, and also in the Pneumonia occurring in old people, and in most cases in children. A very rapid pulse contra-indicates its use, and it is highly dangerous in most forms of the delirium accompanying the disease.

Calomel, with or without opium in combination, has also fallen into disuse, probably not without reason. Experience has gradually demonstrated the minor degree of power which it was at one time supposed to possess in aiding the absorption of exudations, and no valid proof has been afforded that the duration of Pneumonia has been shortened by its use. By most of its advocates it was only held to be efficacious after the previous employment of venesection and the administration of tartar emetic; and a remedy requiring such antecedents is one that may with advantage be abandoned. Even when resolution is delayed, the final termination of the disease is not, in most cases, less favourable; and I should not only feel extremely sceptical as to the value of mercurials in accelerating this process, but I should greatly hesitate to interfere with a remedy which often so materially impairs the general health and nutrition of the patient.[1]

Iodide of Potassium has also appeared to me to exercise little or no influence in promoting resolution.

The methods of treatment by *alkalies*,[2] or by *acetate of lead*,[3] *copper*,[4] and *chloroform*,[5] introduced in more recent periods, only serve to show that Pneumonia is a disease little influenced by remedies; that the less "active" these are the better for the patient. Chloroform inhalations may certainly relieve the cough and allay the discomfort of the patient, as Dr. Walshe has stated, but they appear to have no influence on the progress of the disease.

Digitalis, which was used by Rasori, has recently had an extensive trial, both by Thomas[6] and Ziemssen.[7] This remedy, from the researches of Traube[8] and Wunderlich,[9] seems to have a distinct efficacy in reducing the pyrexia in typhoid fever. It would appear, however, from Thomas's observations, that at periods antecedent to the crisis

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[1] Wittich has published a series of twenty-three cases thus treated, and without fatal results. (Canstatt's Jahresb. 1850.)

[2] Mascagni, quoted by Grisolle.

[3] Leudet, Bull. Thérap. 1863, a mortality of 7 per cent.

[4] Kissel, Canstatt's Jahresb., 1852, a mortality of 4 per cent. All Kissel's cases do not appear to have been thus treated.

[5] Baumgartner, Wucherer, and Helbing, Canstatt's Jahresb. 1850; Varrentrapp, Henle and Pfeufer's Zeitsch. N.F., 1851, analysed in Schmidt's Jahrbucher, lxxiii. 20. The treatment in some of these cases was mixed.

[6] Arch. der Heilk. 1865.

[7] Loc. cit.

[8] Annalen der Charité, i. 691.

[9] Arch. der Heilk. iii.

(except in a few cases, when a marked lowering of the temperature and of the frequency of the pulse is observed), this effect is much less distinct in Pneumonia, but when given in the later stages it tends to increase the post-critical fall to an abnormal degree.[1] Both in adults and children it produces at times intermittence of the pulse, which, however, Ziemssen regards as not intrinsically dangerous. Duclos and Hirtz,[2] who have also used it, give the alcoholic extract in divided doses to the extent of 3, 6, or 10 grains daily. Ziemssen gives ʒj. of an infusion made with gr. v. to the ounce of water every two hours (the infusion of the British Pharmacopœia is made with gr. iij. to the ounce of water).

Veratria, introduced by Aran,[3] has been tested by several subsequent observers[4] with varying results. A more extensive trial of this remedy by Kocher[5] appears to show that in certain cases favourable results may attend its administration in diminishing both the pyrexia and also the frequency of the pulse. In some instances the temperature may be reduced by its use to the normal standard, though in many instances this effect is only temporary, but lasting in others for sixteen hours. In some, however, it appeared to accelerate the period of the crisis, and Kocher is of opinion that it also shortens the duration of the process of resolution; while in a few cases, when given early, it appeared to cut short the disease, and to prevent the occurrence of consolidation. The temperature is commonly affected before the pulse, but in a few cases these phenomena did not coincide; and either the pulse or the temperature may be affected singly and without any corresponding reduction in the other.

The remedy, however, appears in some cases to cause both vomiting and diarrhœa, and to produce, when given in the later stages, a dangerous amount of depression. For this reason Kocher recommends that its effect should be most closely watched, and it appears also desirable that it should only be given in the earliest periods of the disease. The veratria, as an alkaloid, can only be safely given in doses of one-twentieth of a grain, and should be administered in pill, the resin in doses of gr. ⅙.[6] Kocher recommends that it should be given in frequent doses at intervals of from one to two hours, until a distinct effect has been produced upon the pulse and temperature.

In very severe cases he considers that its good effect is increased by venesection. Dessauer, however,[7] who has also used this remedy and

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[1] The effect on the pulse also appears to be uncertain, and a marked lowering of the pulse may ensue without any fall of temperature, though the latter is never observed without the former. Occasionally the reverse effect is observed, and great acceleration of pulse may take place with or without a *rise* of temperature.
[2] Bull. Thérap. vols. li. and lxii.   [3] Ibid. xlv.
[4] Vogt, Schweiz Monatsch. vi., and Bull. Thérap. 58 ; Fournier, Union Méd., 1855 ; Roth, Würzb. Med. Zeitsch. iii. 1863 ; Uhle, Arch. der Heilk. N.F., iii.
[5] Die Behandlung der Croüposen Pneumonie mit Veratrum Preparaten. Würzburg, 1866.
[6] Kocher has found that the tincture of the veratrum viride contains very variable amounts of the alkaloid veratria.
[7] Oesterreich. Zeit. Prakt. Heilk. and Schmidt's Jahresb. 1866, cxxxii.

speaks highly of its effects, regards venesection as unnecessary, and believes that veratria is a complete substitute for bleeding. He considers that no prejudicial effects attend the diarrhœa which it commonly produces, and he says that delirium usually disappears under its influence.

Aconite as a remedy does not appear to have been tested sufficiently to afford a proof of its effects in Pneumonia. In one or two cases in which I have given it I could not observe that any effect was produced by it on the temperature.

The treatment which has hitherto been shown to have the most marked effect on the pyrexia consists in *the external application of cold water to the body.* Tepid baths had been indeed, as Grisolle shows, recommended by Hippocrates, and used by others; and Grisolle himself speaks favourably of their effects in relieving pain and also the general distress of the patient. The use of cold water, though recommended by Currie in fevers, does not appear to have been employed by him in Pneumonia, but it has been largely used by the followers of Preissnitz.[1] It was further introduced into modern practice by Dr. F. Weber,[2] of Kiel, and has been highly praised by Ziemssen, both in the lobar and lobular Pneumonia of children, and by Niemeyer[3] in that of adults. Its effect during the pyrexial period only lasts during, or for a short time after, its employment, and it often requires a prolonged application to effect any marked lowering of the temperature. The reduction of the temperature also by this means appears from Ziemssen's observations to be rarely so marked as in the form of Broncho-pneumonia, and seldom appears to exceed $1\frac{1}{2}°$ or $2°$ Fahr. It appears, however, simultaneously to reduce the frequency of the pulse and of the respiration; and though often unpleasant at first, it seldom fails to afford great relief to the patient, and to produce quiet sleep. The method adopted by Niemeyer is that recommended by Weber, of applying compresses wrung out of cold water, and changed every five minutes, to the chest, and especially to the affected side. Ziemssen recommends the employment of Esmarch's ice-bag,[4] covered with linen, for the same purpose.

In a few cases in children this treatment appears, as also in the form of Broncho-pneumonia, to produce a depressing effect, and it therefore requires to be carefully watched, but it does not appear to be attended with any other risk, either of exciting bronchitis or of setting up

[1] Schedel, quoted by Grisolle.

[2] Beitrage zur Path. Anat. der Neugeborenen, ii. 63. Weber says that this method was first recommended to him in 1837 by Dr. Niessen, of Altona. Grisolle, p. 678, says that it was also recommended by Dr. Campagnano, of Naples, who revived patients in *extremis* by cold baths. Grisolle states that Campagnano also employed bleeding and antimony "*avec une vigueur presque barbare.*"

[3] Spec. Path. Therap. i. 182. Niemeyer states that the treatment has been most extensively used in Prague, with good results. He says that under this treatment cases of Pneumonia rarely last beyond the seventh day; that in an extraordinary number the disease terminates on the third day.

[4] Langenbeck's Archiv für Chirurgie, ii. 275.

secondary complications.[1] It does not appear to shorten the duration of the disease, but only to act beneficially by diminishing the pyrexia.

Blisters in the earlier stages of Pneumonia are to be considered as both useless and as greatly increasing the distress of the patient. When resolution is progressing favourably, they also appear to be quite unnecessary. In a few cases when resolution is delayed, or when there is evidence of a small amount of pleuritic effusion, they may, I believe, in adults be occasionally employed with apparent advantage. In children they are almost invariably inapplicable. Warm fomentations or poultices to the side often give great relief to the pain. I have by no means satisfied myself that any advantage accrues during the acuter stages from any more irritant applications, whether of mustard or turpentine, though in cases of threatening collapse or when dyspnœa is severe, they have occasionally appeared to afford relief.

It may, however, be desirable that after the foregoing analysis some account should be given of the treatment of Pneumonia which is most in accordance with the result of modern observation.

The author, in commencing this branch of the subject, feels it right to express his conviction that a large number of the milder cases occurring in young adults, require no more medicinal interference than similar cases of other acute febrile disorders, and that neither depletory measures nor alcoholic stimulants are necessary to bring such cases to a satisfactory termination.

Rest in bed ; a free supply of fresh, but not too cold, air ;[2] attention to the evacuations, and the administration of a sufficient amount of liquid, nutritious, and easily digestible food—indications abundantly fulfilled by milk and beef-tea—are often all that is sufficient. Pain may be assuaged if severe by a few leeches to the side, by linseed poultices, and more effectually by the hypodermic injection of morphia. Sleep also may be procured by the same means, or by moderate doses of opiates, or probably by the hydrate of chloral.[3] When cough is distressing, and opium is not contra-indicated by cyanosis, this remedy in small doses has appeared to me to give much relief, and to have no injurious effects. Neutral salines also favour the action of the skin, and thus reduce the discomfort from the pyrexia, and probably aid in the elimination of effete matters by the urine. If any

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[1] I have employed this treatment experimentally in only one mild case in a child for a few hours. The continuous application of cold cloths to the chest lowered the temperature half a degree Fahrenheit. It rose again with the ensuing exacerbation to the same height as on the previous evening (103°), after they were discontinued by the nurse, on account of the dislike of the patient to the treatment.

[2] "A close narrow stifling room is exceedingly incommodious to any person sick of a fever, but much more so to those ill of a peripneumony, as I have many times observed, especially among the lower part of tradesmen when two or three families perhaps live in a house together. Celsus' advice is never more proper, nay necessary, in any kind of fever than in a peripneumonia, *in amplo conclavi tenendus æger*. If such close rooms cannot be avoided, they certainly should be frequently but prudently aired." (Huxham on Fevers, 1757, 199.)

[3] I have not had a full opportunity of experimenting with this remedy in Pneumonia.

extensive bronchitis be present, ammonia may with advantage be combined with these, and small doses of ipecacuanha have also under these circumstances appeared to me to be useful. When convalescence is established, solid food and a moderate use of stimulants adapted to the strength and habits of the patient, are frequently all that is necessary to promote a rapid cure. Iron and quinine or strychnia are, however, to be given if there be anæmia or much weakness remaining.

In severe cases of Pneumonia, threatening to invade a large tract of lung, and coming under observation within the first forty-eight or seventy-two hours of the disease, and if the dyspnœa threatens asphyxia, and the distension of the superficial veins indicates overfilling of the right side of the heart, a cautious bleeding may probably be practised with advantage to the extent of six or eight ounces, particularly if the patient be young and vigorous, and of previously temperate habits.[1] Under these circumstances also, if the fever be high, tartar emetic may be given in doses of gr. $\frac{1}{3}$ to gr. j. or gr. iss., combined with salines and small doses of paregoric, every hour or two hours until some relief is experienced—a relief which may be further aided by the application of leeches or cupping to the side. I think it right, however, to add here, that although I have not hitherto adopted the application of cold water in such cases, I should, after the testimony adduced in its favour by the authors before quoted, feel strongly disposed to make a trial of its effects.

Under all circumstances food must be given in suitable quantities, for it is important to husband the resources of the patient as much as possible.

Cases such as these now under consideration vary much in their later manifestations, and it is in these that judgment and decision are most required.

One complication which may be regarded as most indicative of danger is *delirium*, and it is to this symptom especially that I now refer.

By many of Dr. Todd's pupils the occurrence of delirium in Pneumonia has been regarded as a certain indication for the administration of stimulants, and I believe that in the majority of instances the practice is both well founded and successful. Cases do, however,

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[1] Huss lays down the following rules :—Venesection may be practised when the pulse is full, tense, or depressed. The large full pulse sinks at first, but venesection is to be continued until it rises again. In patients with a " tense " pulse venesection is to be continued until it becomes soft. If the pulse is depressed, venesection is to be continued until it becomes full. The indications for venesection to be drawn from the pulse were repeated by nearly every writer of the early part of the present century. How little these were to be relied upon, even by those in the habit of testing their practice by this means, is apparent from the following observations of Hourmann and Dechambre, who may at least be supposed to have been conversant with the fallacy of " fulness " in the pulse of old people to whom these remarks refer : "Nous avons vu des malades chez qui le pouls *invitait* la saignée, cesser de rendre leurs crachats immédiatement après que celle-ci avait été pratiquée et mourir en moins de douze à quinze heures." (Arch. Gén. de Méd., 2e Sér. xii. 190.) Intense severity of dyspnœa appears to me to be the only positive indication for this remedy. A very high amount of pyrexia in the early stages is also so, but to a less degree.

occasionally occur when acute delirium associated with a considerable degree of pyrexia is not benefited by this treatment, and, though comparatively rare, they belong to a class which requires separate consideration.

We have unfortunately but little exact knowledge of the state of the brain during delirium to serve as a pathological guide for its treatment. It is now pretty generally admitted that delirium in many cases is by no means an expression of hyperæmia or inflammatory irritation of the brain, and it is only clinical experience which has led us to the discrimination of these conditions in the various diseases associated with this symptom.

In Pneumonia the evidences, as before stated, of meningeal or cerebral hyperæmia associated with delirium are very rarely met with *post mortem ;* but I believe that we may with advantage discriminate two conditions under which delirium occurs in this disease. In one the state is that of weakness, for which we have no more precise pathological expression ; in the other it is the expression of a blood-poisoning by the products of the pyrexial disturbance, though not, I believe, as some are disposed to think, depending on the direct effects of overheated blood on the nervous centres. It is probable also that in many cases both these conditions are more or less combined in various degrees.

In conditions of pure weakness the reasons for giving stimulants are abundantly clear, but in delirium from blood-poisoning this is more doubtful. It is, however, by no means easy to apply any certain clinical test to distinguish these two states. Delirium with high pyrexia should always induce doubt as to its nature, and this doubt is increased when it has been preceded by severe cephalalgia. I do not think that the special characters of the delirium always afford a certain guide ; at least its violence is no proof of the sthenic or asthenic character of the primary disease, though a low muttering delirium almost invariably belongs to the latter class. A correct opinion on this point must depend on the practitioner's judgment as to the state of the patient's strength ; and if indications of asthenia exist, it is better to depend on this as a guide, rather than on any theoretical reasoning respecting the origin of the symptom.

The state of the pulse is, I believe, the surest indication which we at present possess. An extremely rapid pulse, *i.e.* one above 120 or 130, generally calls for the employment of stimulants. When the pulse presents the characters of dichrotism to any distinct degree, they are almost invariably necessary, and under both these conditions the use of bleeding or tartar emetic is absolutely contra-indicated. Tremors and subsultus rarely co-exist with violent delirium ; when they are present, they also strongly require the remedies under discussion.

In doubtful cases it is safer to make a cautious trial of stimulants than to omit their use : when beneficial, their good effect is usually seen early.

Huss recommends the use of tartar emetic in doses of gr. j. to gr. ij. every hour in the delirium of drunkards, when this sets in early, accompanied by high fever and by a flushed face and tense pulse. He considers bleeding in these cases to be entirely inadmissible, and the tartar emetic is to be discontinued directly the pulse falls in volume, or if diarrhœa or vomiting should occur. The use of all lowering remedies directed solely to the delirium is, however, only to be pursued with the greatest caution, for the diagnosis of the pathological state present is often doubtful, and their danger, when inappropriately used, can hardly be overrated.

Opium in these forms of delirium can only be used with caution. Full doses often increase the prostration, and fail to procure sleep. Huss regards the condition of the pupil as affording a valuable indication for the treatment to be pursued. If this be contracted, opium is contra-indicated, but belladonna, in doses of gr. $\frac{1}{8}$ of the extract, given three or four times daily, may induce a quieter condition, ending in sleep.

I believe that in such cases as these the value of cold applications in lessening pyrexia will be found to be very considerable when properly used, and may aid in solving the difficulty which has hitherto attended some of these cases. Digitalis or veratria,[1] when the pulse is rapid, are remedies that appear to me to be deserving of a further trial than I have yet had opportunities for making of their efficacy.

The class of cases which have now been considered are fortunately comparatively rare. In the majority the discrimination is more simple, and in the severer cases of Pneumonia, the administration of stimulants in the later stages is almost invariably both useful and necessary. They are, indeed, often required almost from the outset in cases marked by debility, at whatever age, but particularly in patients of bad constitution, in those who have indulged freely in alcohol, and in old people; and under all these circumstances attention must be paid to the previous habits of the patient in regulating the amount given.

In such cases as these I believe that all depletion and the use of tartar emetic are in the highest degree injurious, though simple salines may usually be given with apparent advantage.

In the majority of cases the amount of stimulants given during the pyrexial period may be very moderate. It is indeed always best to begin with a minimum dose, and to increase the quantity as required; and under all circumstances it is desirable, as far as possible, to husband resources of this nature. For infants, brandy, which is the best form of alcoholic stimulant for these purposes, may be given

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[1] The lowering of the pulse by veratria is often very considerable. I have known it reduced in acute rheumatism from 100 to 54 in the minute within eight hours by the tincture of the veratrum viride, given in doses of ♏ v. every two hours. The influence of this remedy on the temperature (104°) in this case was much less perceptible. It fell half a degree, and the ensuing exacerbation did not take place. The pulse regained its former frequency within twelve hours after the remedy was discontinued.

in doses of five to ten drops, increased to thirty drops, or ℥j. every two, three, or four hours. For adults, from one to three drachms may be given at similar intervals, and in a large number of cases it is seldom necessary to give more than six or eight ounces of brandy in this manner in the twenty-four hours. The indications for the amount and frequency of these doses are best gained from the pulse and from the general signs of asthenia. As long as these are distinct, stimulants must be persevered with; and though always to be used with caution, they must in some cases, especially in patients addicted to habits of intoxication, be given both unflinchingly and unsparingly when the need arises. I have in one or two instances given 36 ounces of brandy daily for several days consecutively, in doses of six drachms every half-hour, with a successful result, in cases of Pneumonia in drunkards; every attempt to diminish the dose being immediately marked by dangerously increasing signs of asthenia; and it was only when the more marked evidences of prostration diminished, that any symptoms of alcoholic intoxication were observable.

Such cases are, however, rare, and, as before observed, much smaller amounts of alcohol are usually sufficient.

The period immediately following the crisis is that in which moderate doses of alcohol appear to be most called for; and in many cases which have not previously presented marked signs of asthenia, very considerable prostration, which in old people may prove fatal, may occur at this time. Indeed I believe that one of the chief duties of the practitioner in most cases of Pneumonia is to watch carefully for symptoms indicating the employment of stimulants, and to regulate by frequent observations the amount necessary to maintain the strength.[1]

In cases of extreme prostration with a very rapid pulse, and attended by profuse sweating, I believe from what I have seen of the effects of digitalis in the analogous condition of delirium tremens, that this remedy may probably be tried with advantage.[2]

If in the later stages of the disease expectoration becomes profuse

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[1] It is due to the memory of the late Dr. Todd to point out that a great part of the reform in medical practice with respect to the administration of stimulants in acute diseases is due to him. It is possible that he may have pushed this method at times to an extreme, but of their general utility and of the advantage of administering them in repeated doses, as recommended by him, there can now be but little question. It is beyond the scope of this article to enter upon the rather wide discussion to which this practice has given rise respecting the mode of action of this class of remedies. The chemical side of the question will be found discussed in the researches of Lallemand, Perrin, and Duroy, who maintained that the alcohol so given was excreted by the kidneys; while Strauch (De demonstratione spiritus vini in corpore ingesti, Diss. Dorpat. 1862), Schulinus (Arch. der Heilk. 1866), Dr. Hall Smith's "Experiments on the Chromic Acid Test for Alcohol" (Brit. and For. Rev., 1861), and Dr. Anstie (Lect. Roy. Coll. Phys., Lancet, 1867, vol. ii.), have shown that this only takes place to a very limited degree. The latest researches on this subject are by Dr. Parkes and Count Wollowicz (Proc. Roy. Soc. xviii. 1870).

[2] I have known it under these circumstances, when combined with the administration of alcohol (though the remedy had previously been freely given), markedly reduce the frequency of the pulse and increase its power, while the sweating ceased within a few hours after it had been commenced. The digitalis was given in doses of ℥j. of the tincture every two hours.

and copious, and abundant fine râles in the lung show the presence of œdema, and if resolution be proceeding but slowly, expectorants may be used with advantage. The muriate of ammonia and senega appear to be the best of these, and carbonate of ammonia may be beneficially combined with them.

Counter-irritation may at this stage often prove useful.

The maintenance of the general strength is, however, of paramount importance; and bark, quinine, the mineral acids, or preparations of iron, will often promote recovery more rapidly than remedies devoted to the special condition of the lung. Strychnia is useful in cases where much nervous prostration is present. The use of cod-liver oil is also often beneficial at this stage.

It remains to treat briefly of some of the attendant circumstances and complications of the disease.

Severe gastric catarrh, with a loaded and furred tongue, and whether attended or not by vomiting, is in adults often benefited by one or two purgative doses of calomel (gr. j to gr. iij), followed by a saline aperient, and this remedy is recommended by most authors for the "bilious" form of the disorder. Mustard poultices may also be applied to the epigastrium if vomiting is troublesome. In children, however, this symptom may depend on cerebral disturbance.

If diarrhœa be present, a few grains of Dover's powder may be combined with the calomel, and the saline should then be omitted. Severe diarrhœa may, however, require the use of astringents, though, as far as I have observed, this symptom is seldom sufficiently intense to call for their employment. Huss recommends cold compresses to the abdomen, or leeching to the colon, in the dysenteric diarrhœa which accompanies Pneumonia in hot seasons.

If gastric catarrh continues in the later stages, simple alkaline remedies, the bicarbonate of soda combined with bismuth, have appeared to me the most useful. Huss and other German authorities recommend the muriate of ammonia for this symptom.

Hæmoptysis, if profuse, may be met by the internal administration of styptics. The most efficacious of these will probably be found to be gallic acid, acetate of lead, and ergot. The latter is especially recommended by Huss when the pulse is quick, small, and weak. Venesection has been recommended for this symptom, but its true efficacy may be considered as doubtful. It must be remembered that large hæmoptysis is most commonly a symptom of attendant tubercles, and that any reducing measures are, in such a case, specially contra-indicated.

For the condition of *grey hepatization,* Huss and Grisolle recommend the use of camphor, musk, and turpentine. It must be remembered, however, that the full employment of stimulants does not appear to have been practised by these authors. Their administration appears to me to be likely to be better than that of the remedies in question; though these, of which however I have no experience, may at times be useful. Huss recommends the oil of turpentine in doses of five to

ten drops every two hours, and says that it is particularly valuable in the Pneumonia occurring in the course of typhoid fever. He remarks that it seldom disagrees even when the tongue is dry and coated, but that if it causes vomiting it may be combined with hydrocyanic acid. He recommends camphor when delirium is present. This remedy, however, appears occasionally to produce redness of the face and dryness of the skin, and under these circumstances it is to be replaced by ammonia.

For the complication of *abscess of the lung*, Huss recommends acetate of lead in doses of gr. ij repeated every four or six hours, as long as the sputa continue offensive and copious. In the later stages bark or quinine with the mineral acids (Huss considers the phosphoric acid to be the best) are the most suitable remedies.

Gangrene of the lung appears to be but little open to remedial treatment. The employment of inhalations of turpentine, recommended by Skoda, or of chloroform, has proved useless in Huss's experience. Two cases recovered in his hands; one under the internal administration of creosote in doses of one drop given every two hours, and another with pyroxylic acid in doses of ten drops, combined with five drops of tinct. opii every two hours, but the same remedies proved ineffectual in other cases. More reliance must probably be placed both in this and in the last-named condition, on the maintenance of the strength of the patient by abundant support, and by bark and ammonia or the mineral acids.

Pneumonia complicated by *intermittent fever* requires the use of quinine. Huss recommends that eight grains should be given during the rigor, and repeated in the sweating stage.

The complication with *pre-existing Bright's disease* also calls in Huss's opinion for the use of turpentine. I have no experience of this method of treatment. It might, however, prove valuable if alcohol appeared inadmissible in such cases. Huss does not appear to regard this remedy as productive of injurious effects on the condition of the kidneys.

For the complication with *pericarditis*, local cupping or leeching and the internal administration of mercurials have been recommended. The utility of all these measures is, however, I believe, in the highest degree doubtful. Deaths from Pneumonia complicated with pericarditis have always appeared to me to present the most marked symptoms of asthenia. The advisability of small local bleedings must, however, be considered in relation to the general strength of the patient.

For *œdema of a limb* remaining after the disappearance of the disease, friction, shampooing, and an elastic bandage are the most appropriate remedies. (Walshe.)

PNEUMONIA, when appearing as secondary to other dise
some cases both the anatomical and the clinical featu
primary form.   In other instances the disease appe
variable size irregularly scattered through the lung
received the name of Lobular Pneumonia, though
strictly limited to individual lobules as this name wo

The features of the disease, when of the latter class,
when occurring in children, differ so widely from the
to require a separate description.

A short account will also be given of the principal v
characters of Pneumonia when appearing as a compl
disorders.

"CATARRHAL PNEUMONIA" is a variety of Pneumo
racters are in some respects clinically, and in others
only imperfectly defined from those of the acute prima

Until recently it has been considered to be almos
disease of childhood, originating either in primary l
the bronchitis secondary to measles, hooping-cough, an
in some cases of diphtheria.   It is probable, however,
of the pneumonia of old age may belong more truly to
and some recent German authorities have been dispo
logical considerations—which appear, however, to th
on insufficient foundations—to regard many other cases,
with the primary disease, as belonging to this variety
Pneumonia is almost constantly characterised by bei
catarrh of the bronchial mucous membrane ; and it is a
complication of bronchial dilatation.   The inflammat
cular structure of the lungs is in such cases the result
extension of the inflammatory process, or it is induc

which, without the intervention of other causes, would not have led to the inflammation of the pulmonary tissue. In both these classes of cases the invasion of the pneumonia is sudden—it runs a typical course, and terminates by a crisis within the usual period.[1]

In a third class, however, which may truly be termed Broncho-Pneumonia, the invasion is gradual; it is preceded by bronchitis of some standing or intensity, and the implication of the pulmonary tissue is only marked by an increased pyrexia, or by a slight sense of chilliness, usually without rigors, and by prostration with a quick and small pulse and a tendency to sub-delirium, sometimes attended by, but at others without, distinct changes in the characters of the cough and sputa. The latter are usually bronchitic throughout, or they may be puriform, and in a certain proportion of cases rusty sputa are observed. The course of the disease in these cases is protracted and indefinite, either ending fatally, or by a slow lysis and very gradual resolution. In fatal cases the lung is very commonly found in a state of grey hepatization. In a few cases again the invasion may be insidious and gradual, attended by cough and by increasing weakness, but the symptoms may be of such slight comparative severity that patients so affected may continue during some weeks, although with difficulty, their usual occupations. Cases of this class, which bear a strong resemblance to the variety described as "Latent Pneumonia," tend to pass into chronic forms of the disease; and, though occasionally occurring without the complication of tubercles, they have appeared to me, in most instances, to be more or less closely associated with this diathesis.

This form of Pneumonia is, however, common during epidemics of influenza, but it may occur without the direct effect of this specific poison. Huss met with it in 140 out of 2,616 cases, or in a proportion to all forms of Pneumonia of about $\frac{1}{19}$. The mortality is, however, greater than that of the acute primary form, amounting to 14·28 per cent. It is also very common in tuberculosis, of which it forms a most dangerous complication, and markedly hastens the fatal issue. This association and the clinical phenomena attending it belong, however, more properly to the subject of Phthisis, and will not therefore be considered here.

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[1] Out of fifty-three cases I found thirteen to have been preceded by catarrh. In four of these the cough preceded the rigor from twenty-four to seventy-two hours; in one, a chill had taken place a week before the rigor. In three there had been cough for a week before the sudden invasion of the Pneumonia, which commenced either with rigors or vomiting. In three others there was a history of chronic bronchitis. In all these the invasion of the Pneumonia was sudden : two of these cases died on the seventh and eighth days. Of these the affected lung was in one in a state of typical red hepatization ; in the other, in a state of grey hepatization. In one case there was a history of previous catarrh of indefinite duration ; the invasion was sudden, but] the case was protracted. In one only was the invasion gradual. It was, however, a distinct case of Acute Pneumonia.

BRONCHO-PNEUMONIA ;[1] LOBULAR,[2] DISSEMINATED, OR VESICULAR PNEUMONIA.

THE Broncho-Pneumonia of childhood was by earlier writers largely confounded with collapse of the lung, which was considered a result of inflammation before Legendre and Bailly demonstrated its true character. The publication of their observations led indeed to an almost equally strong reaction in the opposite direction, and it has been thought by many that no true infantile Pneumonia ever accompanies bronchial catarrh, but that all the changes in the lung attending this state are due to collapse alone. This opinion, however, is almost equally erroneous with that which it has displaced, since both pathologically and clinically, inflammation affecting the pulmonary tissue has, under these circumstances, certain well-marked features which it is important to recognise.

The peculiarity of this form of disease consists, as before stated, in its origin in pre-existing bronchial catarrh, either extending from the upper air-passages or commencing as capillary bronchitis. It is not, however, always easy to decide the precise period at which the extension of the disease from the bronchi to the air-vesicles takes place, since this is usually gradual, and in scattered points ; and hence in some cases, in children, the condition of Broncho-Pneumonia represents a variable combination of bronchitis and of Vesicular Pneumonia, the symptoms of which are also in part due to attendant collapse.

For the proper understanding of its clinical features and physical signs it is necessary, however, to anticipate so far the description to be hereafter given of its morbid anatomy by stating that the mode of implication of the pulmonary tissue ordinarily differs from that found in the Acute Primary or Lobar Pneumonia, and that the nodules of pneumonic consolidation are usually scattered through tracts of air-containing tissue, which is often emphysematous ; that these nodules may vary in size from the dimensions of a poppy-seed to those of a walnut, and that they may coalesce until larger tracts are invaded ; and further, that the inflammatory changes often commence in portions of collapsed lung ; and finally that both lungs are very frequently and simultaneously affected.

ETIOLOGY.—The frequency of this form of Pneumonia in children is variously stated. Ziemssen[3] observed 98 cases as contrasted with 186 of the primary form. Steffen,[4] for 94 of the primary, has met with 72 of the catarrhal or lobular form.

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[1] The term first used by Seiffert (Die Broncho-Pneumonie der Neugeborenen und Säuglinge, 1837).
[2] Burnet ; Journ. Hebdomadaire, 1833.
[3] Loc. cit.   [4] Klinik der Kinderkrankheiten.

It is most common and most fatal in the earlier periods of life. Of 72 cases observed by Steffen, 52 occurred before four years of age. The age thus specially prone to it corresponds, therefore, with the period of the first dentition; but whether any increased liability to the disease is induced by this process appears to be doubtful, since it is almost constantly a secondary effect of bronchitis, or of diseases of which bronchitis is a common complication in early life.[1] The causes of bronchitis in children are therefore in some degree also causes of Broncho-Pneumonia, and hence it is most common in cold seasons. It is also said to occur at times epidemically, but this is probably due in great measure to the prevalence of influenza or of other zymotic diseases associated with bronchial catarrh.

There appears to be little doubt that a previous condition of bad nutrition markedly predisposes to this form of Pneumonia. The influence of bad air has also been strongly insisted on by Bartels as a more or less direct cause of its occurrence in cases of measles. It is probable also that all causes which diminish the respiratory muscular force of children operate in the same direction, particularly when it is remembered that the occurrence of partial or general collapse is frequently the immediate precursor of the inflammatory changes, and that a long-maintained recumbent position, by causing congestion of the posterior portions of the lungs, favours the pneumonic process. Constitutional predisposition to bronchitis at early ages also favours the occurrence of this disease, and thus it is prone to recur in the same individual.

[1] Steiner (Prager Vierteljahresch. 1862, vol. lxxv.) gives the following table of conditions coincident with Lobular Pneumonia :—

	Cases.	Boys.	Girls.
Rickets	26	15	11
Rickets and tubercle combined	11	8	3
Atrophy	16	10	6
Tubercle of glands	15	9	6
Measles	10	7	3
Scarlatina	3	2	1
Small-pox	2	1	1
Dysentery	7	5	2
Noma	2	2	0
Heart-disease	1	1	0
Meningitis	1	1	0
Burns	1	0	1

Steffen, out of fifty-two cases, found thirty-eight arising from bronchitis, eight from hooping-cough, and six from measles.

Ziemssen, in ninety-eight cases, found thirty-two associated with bronchitis or chronic bronchitis, twenty-three with hooping-cough, and forty-three with measles.

Bartels (Virchow's Archiv, xxi. p. 75) found in an epidemic of measles that 12 per cent. of those attacked were affected with Broncho-Pneumonia.

Peter (Gaz. Hebdom. 1863, p. 689) found the disease very frequent in diphtheria. He states that in 100 cases of diphtheria he found sixty-seven of confirmed Pneumonia and twelve of engorgement. Peter's data would appear, however, somewhat to overrate this frequency, for in some of the cases which he cites as instances of "hepatization," the portions affected floated in water; and he also speaks of collapse as a stage of Pneumonia.

Dr. Wilks (Guy's Hosp. Rep. 3rd Ser. vi. 146) finds that burns of the skin are very frequently followed by this variety of Pneumonia. In some instances, however, it assumed a more extensive and lobar form.

SYMPTOMS.—The signs of pneumonic inflammation are usually developed more acutely in the course of capillary bronchitis and of measles,[1] in which the bronchial inflammation is more intense, than in hooping-cough, in which latter disease the invasion is more gradual, and is almost invariably preceded by pulmonary collapse.

The period of its accession varies also in different diseases. In measles it most commonly occurs during the decline of the eruption, and it may be deferred until the second or even to the third week after the subsidence of the pyrexia and of the exanthem ; occasionally, however, it has been noticed to precede the eruption by a period of nearly a week.[2] In diphtheria the pulmonary complications usually occur within the first five or six days; but in hooping-cough they seldom appear until the disease has been considerably protracted, and both the general nutrition and the muscular power of the patient have been impaired. In acute bronchitis Pneumonia may occur early, when the affection is severe, or when other causes predisposing to pulmonary collapse are present ; and among these, early infancy, rickets, or previously defective nutrition are prominent. In chronic bronchitis the supervention of Pneumonia is commonly a late phenomenon.

The extension of the inflammation from the bronchial mucous membrane to the tissue of the lungs is rarely attended by the phenomena of rigors, or vomiting, or by the cerebral symptoms which mark the invasion of the primary form of the disease, though the latter may occasionally be observed.[3]

The pulmonary complication is usually first evidenced by an increase of dyspnœa, together with the supervention of fever, if this has not been previously present, or by an aggravation of that already existing.

The dyspnœa, except in very mild cases, when it may be comparatively slight in degree, is commonly both objective and subjective, and gives the patient much distress. It tends at times, and particularly in rickets and when collapse of lung is also present, to occur in suffocative paroxysms, and, even when these are less marked, it varies in intensity at different times of the day, and is usually most felt in the morning and evening.

Great acceleration of the respiration is the rule where the disease is of any considerable intensity; it may then equal the extreme degrees of frequency observed in the primary forms of Pneumonia, and may sometimes attain to 107 respirations in the minute.[4] Commonly the frequency of the respiration is, as in the primary disease, dispropor-

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[1] It has, however, been before stated in respect to measles, that some forms of Pneumonia occurring in this disease approximate very closely in their characters to the true primary form, both in the rapidity with which a large tract of lung is invaded, and also in their anatomical characters.

[2] Steffen, loc. cit.

[3] Dr. West (Dis. of Infancy and Childhood, p. 326). Steffen has once seen spurious hydrencephalic symptoms precede the outbreak of the Pneumonia, and cease on its appearance (loc. cit. 302).

[4] Bednär, Lehrbuch der Kinderkrankheiten, 268.

tionately greater than that of the pulse, the ratio of 1 to 1·5 being sometimes observed ; but when cerebral congestion is also present, the pulse may be rapid and the respiration even slower than natural (Bednär). The respiration is not unfrequently retarded when a fatal termination is approaching. Irregularity in its rhythm is not uncommon, and this may amount to so complete a cessation of the respiratory movements during some minutes as even to simulate death.[1]

The thoracic movements are shallow, with great elevation and little expansion. Inspiration is imperfect and short ; expiration is often forcible, prolonged, and noisy. The action of the accessory muscles is violent. The chest is raised by the elevatory muscles, but the lower portions are drawn in by the diaphragm. The anterior superior portions appear distended when emphysema is also present, but may yet be comparatively motionless. The action of the alæ nasi is also greatly exaggerated. The cough varies in character. It is sometimes paroxysmal, but this character, even when previously present, as in hooping-cough, may disappear on the supervention of Pneumonia, and the cough may become short and dry ; and this change in its character often forms, together with the pyrexia, one of the earliest signs of the implication of the pulmonary tissue in this disease. The cough also often becomes painful, eliciting cries from the patient, a symptom which is also sometimes a valuable indication of the pneumonic change. I believe, however, from some cases in which I have observed this in adults, that such pain may be extra-thoracic and myalgic in its nature, and that it is partly caused by the sinking of the inferior parts of the thoracic walls due to collapse of the lung. In some cases the pain complained of may be in the epigastric and in the upper abdominal regions.[2]

The secretion from the bronchi, if previously free, as shown by the looseness of the cough, is often diminished. Expectoration is rarely seen in young children. When brought up by vomiting, it is commonly bronchitic and tenacious, sometimes streaked with blood, but rarely if ever rusty. The same characters are observed in the sputa of adults attacked by Pneumonia during the prevalence of influenza.

The physical signs in the earlier stages are often obscure. They commonly affect both lungs simultaneously, though rarely in an equal degree. The immobility of the thorax and the sinking of the lower ribs, with deepening of the diaphragmatic depression, occur in cases of simple bronchitis, with attendant emphysema and collapse ; but when the lung becomes extensively infiltrated, the depression of the ribs may partially disappear.

The percussion results may be uncertain : usually the upper parts of the chest are hyper-resonant, and they may be quasi-tympanitic when much attendant emphysema is present. When the spots of collapse or of pneumonic infiltration are disseminated through healthy pulmonary tissue, the sound, though less resonant than natural,

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[1] Barthez and Rilliet, i. 264.   [2] Steffen.

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The respiratory sounds over collapsed portions are commonly weak or inaudible. In lobular pneumonic consolidation they usually acquire a bronchial, but never a tubular, character (Walshe). This quality of respiration, though occasionally, is only very rarely met with in simple collapse.[2]

The respiration in other portions of the chest is usually exaggerated and attended by râles. Generally disseminated dry or moist bronchitic râles indicate only the bronchial catarrh. When Pneumonia supervenes they, however, often become finer, and may thus be heard in limited spots of the pulmonary tissue, and they frequently change in site from day to day;[3] but they seldom present the typical characteristics of the crepitation heard in the Acute Lobar Pneumonia. In some cases, however, when the finer bronchi are dilated, the râles heard may be coarse, and they may acquire a quasi-metallic character if consolidation surrounds these dilatations. Râles are seldom heard directly over collapsed parts, unless they be conducted from adjacent bronchi.

Vocal fremitus is commonly exaggerated over pneumonic infiltration more than over collapsed portions of lung. The differences of degree observable in this respect are, however, very variable.

Vocal resonance, as heard when a child cries, is usually much increased by pneumonic consolidation of any extent, and frequently under these circumstances it acquires a bronchophonic tone. These characters may, however, be absent when the bronchi are extensively obstructed.

The pulse is rapid. It rarely, even in the early stages, presents the fulness or strength of the primary disease. At more advanced periods it becomes excessively frequent, small, and feeble, so as scarcely to be felt. Irregularity of its rhythm is also occasionally observed. Fulness of the superficial veins, extending even to those of the hands (Trousseau), is also observed, and œdema of the extremities has sometimes been noted (Steffen).

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Simultaneously with these symptoms there is a great restlessness : the eyes are sunken, and the face assumes an anxious expression, which is painfully distinct in young children. Strength fails rapidly ; as the disease progresses somnolence and a semi-comatose condition supervene, in which the child lies passive, but starting up from time to time into an erect or semi-erect posture, with jactitation and movements of distress, when attacks of cough and dyspnœa return.

The skin is hotter than natural, though not commonly presenting the pungency of heat which characterises the acute lobar form, and it is often bathed in profuse perspiration, which occasionally alternates with a dry heat. The perspiration may be general, or in rickety patients may appear chiefly about the head. The surface is generally pallid with the exception of the cheeks, which present a flushed or violet tinge, which is sometimes transitory and alternates with a cyanotic pallor. Cyanosis of the lips and finger-nails increases with the progress of the disease, and are especially distinct in the pneumonia succeeding to hooping-cough, and when collapse forms a prominent feature.

Vomiting, unless caused by the cough, is less common in this form of Pneumonia than in the acute primary disease. Diarrhœa, on the other hand, is not unfrequent, particularly in the Broncho-Pneumonia attending measles, and if not originally present it is very easily excited by medicinal remedies, especially by tartar emetic. The tongue, at first moist, becomes dry in the later stages, and sordes form on the teeth or on the angles of the lips, which are also dry and cracked ; aphthous stomatitis may occur when the course is protracted. The appetite is completely lost, but thirst is marked ; infants at the breast suck it eagerly, but the power of continued sucking is lost, owing to the difficulty of breathing. A slight degree of delirium is occasionally observed, particularly in older children, as an exaggeration of the restlessness which tends to increase towards night. Convulsions are, however, much less frequent than in the acute disease ; when they do occur, they form a very unfavourable feature. A semi-comatose state is more common ; it passes later into deeper unconsciousness when the signs of mal-oxygenation of the blood become more apparent. In some cases, however, a hydrencephalic condition with restlessness and cries has been observed.[1]

The urine, owing to the early period of life in which the disease usually occurs, has not been made the subject of exact observation. Bednär says that the chlorides are present. In some cases the presence of a small amount of albumen has been noticed.

Emaciation and loss of strength progress with marked rapidity ; there is great loss of weight, the eyes are sunken, the muscles are wasted, and the skin is flaccid. These appearances may, in severe cases, become very distinct within a few days from the outset : if the disease runs a more protracted course, and particularly in the Pneumonia succeeding to hooping-cough, the wasting of the tissues may attain an extreme

[1] Barthez and Rilliet (i. 467) attribute this to frontal neuralgia.

degree of marasmus—proportioned, however, in most cases to the age of the patient and to the severity of the disease. Ecthymatous pustules often form, which lead to painful sores. Excoriations of the nose and angles of the mouth are also observed, and bed-sores form on the prominent parts of the emaciated limbs. The patient often dies completely exhausted, or sinks suddenly during a paroxysm of cough, or with the increasing cyanosis may pass into a state of final somnolence and coma. When death occurs in the early periods of the disease, it is usually due to the combined asphyxiating effects of capillary bronchitis and collapse. The course of the disease after Pneumonia has set in is usually more protracted.

Many of the above symptoms, and especially the increasing intensity of the dyspnœa, may occur in severe cases of bronchitis accompanied by extensive collapse of the lung, and uncomplicated by Pneumonia. The most characteristic feature of the latter is constituted by the pyrexia, the presence of which is almost essential to its recognition. Acute bronchitis in children is, indeed, not unfrequently attended by fever, but when uncomplicated by Pneumonia the temperature seldom rises in it above 101° or 102°. The fever also when present is not continuous, the temperature in the morning being often nearly at the normal standard, or perhaps falling to 99°, or 99·5°. The invasion of Pneumonia is marked by accession of fever if the disease has been previously apyrexial, or by an increased temperature if fever has already existed, and this, in the pneumonia of measles, may speedily attain the degrees of 103°, 104°, or 105°. The lower standard of 102° may, however, not be surpassed in the whole course of the case.[1] Sometimes a rise of temperature may be observed to *follow* the accession of dulness on percussion, both at the outset and during the subsequent exacerbations, showing that collapse has preceded the inflammatory changes. This, however, is not always to be observed, and the rise of temperature may be comparatively sudden and rapid, and may either proceed *pari passu* with the loss of resonance on percussion, or may precede this by some hours or days,—the diminution of pulmonary resonance only becoming distinct when the islets of Lobular Pneumonia, by becoming confluent, affect tracts of tissue sufficiently extensive to give rise to this physical sign.

In some cases again, when Pneumonia succeeds to measles, the invasion both of the physical signs and also of the pyrexia may present a great resemblance to the phenomena of the acute primary disease,—the temperature rising rapidly, and maintaining a tolerably uniform elevation with comparatively slight morning remissions, but in its later periods running a protracted course resembling the catarrhal type.[2] In its subsequent course, however, the pyrexia as measured

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[1] In some fatal cases the temperature may rise shortly before death to upwards of 107° (Ziemssen), but on the other hand a rapid fall of temperature, due probably to defective aëration of the blood and to extension of the collapse, may immediately precede the fatal issue.

[2] Ziemssen and Krabler, Klinische Bericht ueber die Masern und ihre Complicationen, p. 169.

by the temperature presents certain characteristics which aid considerably in the recognition of this form of Pneumonia. The chief among these are the irregular course of the fever, the extent of the remissions and exacerbations, and the absence of critical phenomena, —the fever being usually protracted, and ending only by a slow and gradual decline, which is often interrupted by renewed exacerbations.

The remissions may be as great as from 1·8° to 2·5° Fahr. They occur at irregular times during the day, differing in this respect from the ordinary course of the acute primary disease. It has been already stated that in some cases of this form, the maximum temperature may be observed in the morning instead of at night; but this condition, which is exceptional in Primary Pneumonia, is much more common in Broncho-Pneumonia, when it may be noticed to occur irregularly in the course of a single case,—a peculiarity which is probably due to the indefinite course and irregular extensions of the pulmonary inflammation. The termination of the fever is also protracted. Ziemssen regards a case terminating within seven days as a very exceptional one; and the pyrexia may last for weeks,[1] in the Broncho-Pneumonia both of measles and hooping-cough, presenting in its irregular exacerbations and remissions a striking resemblance to the course of tuberculosis, which, however, according to the observations of Bartels and Ziemssen, is a much less common sequela of these diseases than is usually believed. The defervescence rarely, if ever, presents the abrupt critical fall so commonly observed in the acute primary form; or if this commences, it is usually followed by subsequent elevations of temperature: the decline of the fever is only gradually effected, and rarely extends over a shorter period than three or four days, and it is often interrupted by irregular secondary exacerbations. In some cases the temperature often finally sinks during some hours or days below the normal standard.

The range of temperature is commonly lower, and both the course of the disease and the duration of the pyrexia are more protracted in the Pneumonia succeeding to subacute bronchitis and to hooping-cough than in that which follows measles. In hooping-cough the degree of pyrexia may be very slight, and the morning remissions may attain almost to the normal standard,[2] but exceptional cases occur in which Pneumonia complicating this disease appears in an acute form and runs a rapid course to a fatal termination.

With the gradual decline of temperature other signs of improvement become evident. There is rarely any appearance of critical sweating, but perspiration appears from time to time, and often seems to afford relief. The dyspnœa and the cyanotic aspect diminish; the pulse and respiration fall in frequency; the cough becomes looser and less hard, and it may again acquire a paroxysmal character, if this has, as in

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[1] Eight weeks. (Bartels.)
[2] See a case by Steffen, p. 313. This case was the more remarkable, as it was one of hooping-cough complicated by tuberculosis, which had progressed to the stage of excavation.

hooping-cough, been previously present; diarrhœa, if present, ceases; the appetite gradually returns, and thirst disappears or diminishes in intensity.

Recovery is, however, almost always slow and protracted; cough persists long; and the duration of the physical signs of consolidation, and especially of bronchitic râles, may continue during many weeks. Some acceleration of the respiration and of the pulse may also continue after the fever has subsided.[1] Slight returns of the pyrexial symptoms may also be observed during this period. The restoration of the digestive powers and of the nutrition is only very gradually effected, and the patient may be for months liable to a renewal of catarrh, attended with slight degrees of feverishness.

In some cases, after long continuance of the pyrexia and of the physical signs, the former may subside, but the latter may change their character and present those of chronic pneumonia or of bronchiectasis, or sometimes of the latter alone.[2]

Complications.—Independently of the diseases which exert a direct or predisposing influence on its production, the liability of Broncho-Pneumonia to other complications is comparatively slight. Some of these will be further alluded to among attendant pathological phenomena. One of the most important is intestinal catarrh, which has been attributed by M. Beau to the swallowing of unhealthy sputa. It appears, however, to be more directly due to the venous congestion of the intestinal canal, and to the tendency of catarrh in children to affect the whole gastro-pulmonary tract of mucous membranes. True dysentery was observed by Steiner in seven out of 110 cases. Pleurisy is less frequently observed than in the acute primary disease. Steffen in seventy-two cases only found six of extensive pleuritic effusion. Various degrees of the affection, in the form of adhesions, are by no means uncommon.

Tubercle is not uncommonly associated with the chronic form, but whether as cause or effect it is not in all cases easy to distinguish. It is not improbable that repeated relapses may give rise to this tendency, and a statement of Steiner's, which will be alluded to hereafter, would seem to show that even collapse may suffice to set up tubercular formations in predisposed subjects; and Bartels has remarked that when present it is specially prone to occur in the condensed portions. On the other hand, the statements of Bartels appear to show that tuberculosis is a much less common sequela of the Broncho-Pneumonia of measles than has been commonly believed.[3]

PATHOLOGY AND PATHENOGENESIS.—It has been already stated

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[1] Steffen.

[2] This subject will be again referred to under the head of Chronic Pneumonia.

[3] Bartels only found tubercle four times in twenty-one post-mortem examinations, and in two of these the affection was meningeal. Ziemssen only observed cheesy changes twice in sixty-three cases of death. Legendre and Bailly found five cases of tubercle in twenty-seven of catarrhal Pneumonia. Ziemssen and Krabler remark, however, that acute tuberculosis is not uncommon *after* measles.

that this form of Pneumonia may be produced by the mechanism of two distinct processes. The pulmonary alveoli may suffer by the direct extension of the inflammatory action from the bronchi, or the inflammatory changes may only take place in portions which have already become the subjects of collapse. The pathological appearances in the inflamed lung present under these conditions several points of difference. Under both sets of circumstances the changes in the mucous membrane of the bronchi are very similar, exhibiting various degrees of inflammatory congestion, together with swelling and softening of the mucous membrane, which are accompanied by slight superficial ulcerations. These changes may appear throughout the whole course of the air-passages when the catarrh has commenced in the larynx and trachea and has travelled downwards. In some cases, however, they may be limited to the smaller bronchi, and the upper air-passages may present a comparative immunity. The walls of the finer bronchi are often so much thickened as to cause them to stand out rigidly in sections of the pulmonary tissue.

Dilatations of the bronchi are very common: they are generally in the cylindrical form, but are sometimes globular, and they may be so universal throughout the lung as to give it a cribriform aspect on section, or even to present the appearance of a number of small cavities. The contents of the bronchial tubes are usually a creamy pus, or a denser exudation constituting a form of false membrane, or sometimes a clear tenacious mucus containing nucleated cells in which a large proportion of desquamated ciliated epithelium may be found. In other cases again little or no mucus can be found in them.

The changes in the lung tissue, when the inflammation has proceeded directly from the bronchi, present the appearance of a number of small whitish yellow spots with indistinct margins fading insensibly into the surrounding tissue. They are not very prominent,[1] and do not stand out sharply defined like the grey granulations or the small softer spots of more acutely produced tubercle. They are often very thickly scattered through the pulmonary tissue, and the portions of lung intervening between them are softer, more vascular, and more œdematous than natural. They are slightly but not markedly granular, and usually, on scraping or pressure, a turbid milky fluid can be expressed from them. As they increase in age they become firmer and drier, but still have little of the granular character. That they undergo any cheesy metamorphosis independently of pre-existing or superadded tubercular changes is in my opinion very doubtful. In addition to these spots others are found, varying in size from a pin's point to a hemp-seed, of a brighter yellow, consisting of dilated air-vesicles, or sometimes of groups of air-vesicles whose walls have broken down, or of the terminal extremities of dilated bronchi. In whichever manner originating, they form little collections of puriform fluid, which escapes when they are pricked, but which also may become more

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[1] These spots are often described as "prominent," but the author believes that as a question of degree the distinction here drawn will be found to be correct.

inspissated, and which when evacuated leave irregular cavities. They are sometimes found under the pleura forming little sub-pleural abscesses, and in some cases may by their rupture give rise to pneumothorax. It is doubted by some writers whether these originate in a true inflammation of the air-vesicles of the lungs, or whether they are not merely collections of pus which have gravitated or have been drawn by inspiration into the extremities of the dilated bronchi and infundibula. I believe from my own experiments on animals in which, especially in dogs, both these sets of appearances can be produced easily by injecting ammonia into the trachea,[1] that they are to be regarded as truly inflammatory in their nature; though the latter mode of origin is however possible, since, as Ziemssen (who agrees with the view previously expressed by Fauvel of their nature) remarks, they are most commonly to be found after the prolonged paroxysms of hooping-cough. The purulent yellow spots before described are perhaps more commonly found in the Pneumonia succeeding to measles than in other conditions.

I have before stated that the Pneumonia which is secondary to diphtheria may present in some cases all the anatomical characteristics of the acute primary form. In other cases it takes place by the intervention of collapse; but in a third class the appearances observed correspond with those which have just been described. When the bronchial ramifications are opened it will be found, in proportion as these diminish in size, that the firmness of the exudation layer diminishes, and that the finer bronchi are only filled with a soft puriform fluid. In some instances this process does not extend beyond bronchi of from two to four lines in diameter; but, in other cases, patches of a yellowish colour and soft consistence, varying in size from that of a hemp-seed to a horse-bean, and only rarely attaining the dimensions of a hazel-nut, are found in the pulmonary tissue. These are impervious to air, and are only slightly granular. They fade insensibly into the surrounding tissue by an ill-defined margin. They are friable, and on pressure they allow a thin yellowish fluid to exude. On microscopic examination the air-vesicles are found, for the most part, occupied by an amorphous exudation, in which are seen a few puriform and granular cells, together with proliferating epithelium from the air-vesicles. I agree with Sir William Jenner,[2] that in most cases these spots are formed by the fluid formed in the bronchi being drawn by inspiration into the air-vesicles of the lungs.

In the Pneumonia which occurs consecutively to collapse of the lung,[3] various stages may be observed in the inflammatory process

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[1] See p. 616.   [2] Oral communication.

[3] Hasse (Path. Anat., Syd. Soc. Ed., p. 251) says that Pneumonia is not necessarily frequently associated with atelectasis. This may be true of primary atelectatis in the recently born infant, but I believe that acquired collapse frequently forms the starting-point for secondary Pneumonia. Hasse says that he has seen spots of collapse in the midst of hepatized lung, but not participating in the inflammatory changes. There is no doubt but that Pneumonia and collapse may not unfrequently be found co-existing, but I believe that it is quite as common to find Pneumonia commencing in the midst of

It requires, however, to be stated, that in many fatal cases of bronchitis, and particularly in infants, the condition of extensive collapse, unattended by a trace of inflammation, may be the only morbid change present.

The mechanism of the production of collapse will be more fully treated of in the section devoted to this subject. It is therefore only necessary in this place to refer to the inflammatory changes ensuing in parts which have already undergone this change.

In collapse pure and simple, the parts affected are sunk below the level of the surrounding tissue. They are most usually found at the bases of the lungs and at the free borders of the lower inferior lobe, and they commonly affect both lungs simultaneously, though rarely in an equal degree. They are often pyramidal in shape, with the base at the periphery of the lung corresponding to the distribution of the terminal bronchi leading to the affected parts. They sometimes, however, occur in the more central portions. They are irregularly distributed, and vary in size from that of a hemp-seed when they arise from the collapse of small groups of pulmonary lobules, to spots of one or two inches in diameter. In severe cases, both in infants and in adults, a whole lobe may be affected, though the pyramidal form is that most commonly observed.[1] On section the nodules are of a bluish purple tint, which is uniform except when traversed by bronchi, blood-vessels, or interlobular septa; they are smooth on section, allowing only a small amount of blood-stained serosity to escape on pressure or by scraping; they are often, however, attended with scattered ecchymoses. They are resistant, and do not break down readily under pressure, and they are airless and sink in water, but can be restored to their normal condition of expansion by inflation, which process, however, leaves the affected parts of a brighter red than the surrounding tissue. This latter is, however, more or less congested and œdematous, and not unfrequently emphysematous. When the condition has lasted longer, the ability to insufflate the lung diminishes, and may even be partly lost, and the parts thus affected may finally undergo either a simple atrophy, or may become the seat of fibroid metamorphosis or of calcareous degeneration.[2] These changes belong, however, rather to the pathological history of bronchitis than to that of Pneumonia, with which they have no

collapsed portions, as it is to observe the condition described by Hasse. The elucidation of the origin and nature of collapse, and the recognition of the distinction between this condition and Pneumonia, appear to have led many writers since the publication of the researches of Legendre and Bailly to deny too exclusively the existence of Lobular Pneumonia in children, and to attribute all the appearances of consolidation found in their lungs to the condition of collapse. There is no doubt as to the comparative frequency and the important pathological and clinical significance of this condition, but I believe that these have been to some degree exaggerated, and the importance of pneumonic changes has been in consequence underrated.

[1] In some cases of collapse from pneumo-thorax a whole lung may thus become affected by secondary Pneumonia (Steffen, loc. cit. p. 24).

[2] Hasse (Path. Anat., 250), Gairdner (Brit. and For. Rev., April 1853, p. 467 ; Path. Anat. of Bronchitis, p. 68).

necessary connexion, though under the names of CARNIFICATION and CARNISATION [1] they have often been confounded with it.

The process of inflammation in the collapsed parts is effected through two sets of changes, which, however, differ chiefly through the degree in which they are complicated by passive congestion. In some cases this is extensive, and affects large tracts which have previously become collapsed, and extends also to the surrounding tissue. The cause of this congestion appears to be the increased impediment to the circulation arising from defective aëration of the blood, and also from the absence of the alternate expansions and contractions of the pulmonary tissue, which, under normal conditions, largely favour the passage of blood through the capillaries of the lung. To this passive congestion œdema quickly succeeds. The tissue then loses its bluish tint and becomes of a darker purple; much serosity exudes on section; and this, in the parts not previously collapsed, may be frothy from the admixture of air. The tissue also becomes more swollen, and the tough resistant character of simple collapse being lost, it is also rendered more friable and breaks down under pressure. To this state the term SPLENISATION has been applied, and it is sometimes erroneously confounded with the "Pneumonie de agonisies" of Piorry, though the changes now described are not, in their essential nature, of an inflammatory character. Congestion of this kind may affect large tracts of tissue surrounding collapsed portions, and hence it has been observed that these parts may be crepitant, or that the collapsed and congested portions may be insufflated; and from this fact has arisen the statement that insufflation is possible in the early stages of Lobular Pneumonia. When inflammatory changes occur in such parts they appear generally in scattered nodules which are solid and granular, but very friable, and in which the interlobular septa have disappeared and the ordinary character of collapsed tissue is destroyed. These nodules are whiter in colour than the dark purple tissue surrounding them, but into which they fade insensibly at their margins. They depend on the accumulation, in the interior of the air-vesicles, of enlarged epithelial cells, mucoid cells, and pyoid cells, which fill and distend them; and hence, in addition to being solid, the parts thus affected become prominent above the level of the surrounding tissue. Collapsed portions which have become congested sink in water without pressure. Congested parts surrounding these usually float imperfectly, but sink after pressure. The pneumonic nodules sink without pressure. On scraping the latter a milky fluid exudes, airless, and presenting under the microscope cells of the same character as those found in the pulmonary alveoli. As the process extends, the whole

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[1] It appears best to confine the significance of these terms to simple "collapse," which presents an appearance which they fully describe. The application of them to other conditions only involves confusion. MM. Isambert and Robin (Gaz. Méd. 1855) have applied the title of "Carnification congestive" to a form of induration of the lung secondary to heart disease.

of the collapsed and congested parts may gradually become infil-
trated until the greater part is solidified, but usually the nodular
form is preserved for some time with congested and excessively œde-
matous tissue intervening between the pneumonic portions. This form
of Pneumonia readily passes into the condition of grey hepatization.
The congestion disappears from the infiltrated parts, and the grey
appearance is produced by the rapid progressive fatty degeneration
and liquefaction of the inflammatory products, aided by the co-
existence of œdema.

Pneumonic changes may, however, occur in collapsed portions,
without being preceded by such marked evidences of congestion and
œdema as those last described. Under these circumstances, the ap-
pearances presented offer a considerable resemblance to the first form
of Vesicular Pneumonia, and it may be questioned whether in some
cases the existence of collapse is not an accidental complication super-
added to the pneumonic process, rather than an essential factor in its
production. In the collapsed portions nodules are found, of a greyish
yellow, contrasting strongly with the surrounding purplish tint of the
adjacent tissue. They are also prominent about its level. They
are solid, and usually granular, and yield a milky or yellowish
creamy fluid on scraping or pressure. In them, however, dilated
bronchi filled with a yellowish puriform fluid are often observed, and
these may even give the appearance of small abscesses scattered
through the nodules; when evacuated they leave small cavities. Dilated
bronchi with similar characters may sometimes be found in parts
affected by simple collapse;[1] but unless true Pneumonia be superadded,
the granular character of the adjacent pulmonary tissue is absent.

These appearances may occur in collapsed portions of lung, how-
ever originating. I have found pneumonic nodules identical with
those last described in the base of the lung of an adult dying of uræmic
poisoning, and who had presented during life no signs of bronchitis,
but in whom dulness on percussion, unattended with pyrexia, had
appeared at the base during the later days of life. The whole of the
lower lobe of one lung was collapsed, and in this part were spots
of Pneumonia surrounding small dilated bronchi, which were filled
with a yellowish pus. It has been recognised since the observations of
Dr. Baly,[2] that in the exhausting diseases of adults collapse may
easily occur, probably from muscular weakness, though this change
is comparatively less frequent in them than in children, owing to the
more yielding character of the chest walls and the smaller calibre of
the bronchial tubes in the latter. Collapse is thus in many cases
the first stage in the production of hypostatic Pneumonia, so fre-
quently found in post-mortem observations. Hourmann and Dechambre
had previously described appearances identical with these in the
lungs of the aged, as consisting of scattered yellow or whitish spots,
from which a yellow fluid escaped, leaving behind small vesicles

[1] Dr. Graily Hewitt, "Pathology of Hooping Cough."
[2] Communicated to and cited by Dr. West (Dis. of Infancy and Childhood, p. 291).

situated in the midst of congested tissue,[1] and passing at
into larger nodules of a granular appearance; and Durand-F
has confirmed these observations.

It is not uncommon to find the processes of collapse, conge
and Lobular Pneumonia intermingled in the same lung, and
stage may be occasionally observed between them. The char
of the pneumonic change also vary, and in some parts nodule
be found resembling in all their essential features spots of red
tization, as observed in the acute primary form, while in othe
pneumonic portions may be white or grey, or almost diffluent,
softening into abscesses.

In all the forms now described, and in fact in nearly all cases
nodules of Pneumonia reach the surface of the lung the pleur
ticipates in the inflammatory changes, and a layer of lymph is
on the surface, which is congested and roughened. These cha
may sometimes aid in the distinction between Pneumonia and
collapse, for in the latter, though sub-pleural ecchymoses are son
observed, a true inflammation of the pleura is rarely found. Ex
pleural effusion is, however, very unfrequent in Lobular Pneum

Scattered nodules of red hepatization, occurring apparently w
the intervention of collapse, and also without any appeara
direct extension from the bronchi, are found in other blood di
I have seen them in small-pox[3] and diphtheria, and they als
sionally occur in scarlatina and typhoid fever. It is probab
that some forms of the Pneumonia-occurring in the course of
originate in a similar manner. These cases certainly militate
the view expressed by some recent writers, that the acute p
or croupous Pneumonia is a specific disease with special ana
characters.

All these disseminated varieties of Pneumonia, including th
vesicular or broncho-pneumonia of measles, may occasionally c
and occupy large tracts of the lung,[4] and under these circum
also the anatomical distinctions from the acute primary disease l
very uncertain, though the consolidated tissue is generally pal
whiter, and presents a greater number of puriform spots, arisin
the accumulation of pus in previously dilated bronchi, than ar
monly observed in the acute primary form. In the rarer cases, v
Lobar Pneumonia appears to be produced in adults by a gradual
sion from the bronchi, there are few, if any, exact anatomical ol
tions respecting the condition in the earlier stages. Most of
except in old people, occur in tubercular subjects. A few pass in
condition of Chronic Pneumonia, hereafter to be described. Th

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[1] Arch. Gén., xii. 2ᵉ Sér. 274.
[2] Maladies des Vieillards, 475.
[3] Steffen says that yellow spots corresponding to puriform collections in
bronchi may occasionally be found in the secondary Pneumonia of variola (
p. 294).
[4] The "Pneumonie Lobulaire Généralisée" of Barthez and Rilliet.

ing characteristic of such cases, when non-tubercular, appears to be a tendency to pass early into a state of grey hepatization.

There is also in the typical forms of catarrhal Pneumonia, a greater amount of proliferation of epithelial cells, and a smaller number of pus corpuscles, at least in the earlier stages, than is observed in the primary disease. A spontaneously coagulable fibrinous exudation is rarely present, and the material in which the cells are imbedded is semi-fluid and allied to mucus, approaching in this respect the characters of the gelatinous-looking exudation which fills the air-cells in the pneumonia

FIG. G.

attending many cases of tuberculosis; I believe, however, that all gradations can be observed between these products.[1] In some cases also of the most distinct hypostatic Pneumonia, I have found the air-vesicles filled almost entirely with pyoid cells, resembling those seen in the later stages of the acute primary disease. (See Fig. E, p. 669.)

In the further progress of Broncho-pneumonia there is little doubt but that in many cases a perfect *restitutio ad integrum* may occur, and that the lung may regain its normal condition. In other instances, however, dilated bronchi may persist long with some condensation of the pulmonary tissue surrounding them, but may gradually return to the normal state, as far as may be judged of from the physical signs. Abscesses also may occasionally form, though usually they are small in size, and gangrene is also sometimes observed in the affected portions.[2] In rarer instances, general consolidation of the lung may remain in a chronic form, the characters of which will be described under the head of Chronic Pneumonia. In some instances the pneumonic nodules, particularly in scrofulous and rickety children, may pass into cheesy changes with destruction of tissue, and may run the subsequent course of tubercle. Ziemssen describes these as a true tuber-

[1] See Appendix D.
[2] This is, however, rare. Steiner only observed two cases. Steffen also reports two such. It is rather more common in the indurations surrounding chronic bronchial dilatations.

culization. Barthez and Rilliet [1] also describe them as surround
a zone of grey induration, precisely resembling in this respe
changes found around tubercles in the adult. Bartels, as a
stated, has found tubercle to be a less common complication
pneumonia of measles than is commonly believed; but I ar
vinced, from my own microscopic observations, that tubercle may
in these spots of pneumonic change, although it may be maske
may be undiscoverable by mere ocular inspection.[2] The pneu
changes are always limited to an accumulation of cells in the i
of the air-vesicles and terminal bronchi, and, except in very
forms, when interstitial thickening occurs, the walls of the
themselves are not the seat of nuclear growth.

The associated pathology of Broncho-pneumonia presents b
special features independently of those of the diseases of which
stitutes a complication. The dilatation of the right side of th
resulting from obstruction to the pulmonary circulation, may
permanence of the openings of the foramen ovale and ductus arte
Thrombosis of the pulmonary artery is occasionally observed.
carditis is also an occasional complication. The bronchial gla
swollen and medullary-looking. Sometimes they are distinctly
æmic, but when the swelling is extreme they may be pale. I
cases they are unaffected. Sometimes cheesy spots or calci
dules are found in them, but these usually accompany tubercle
lung. Suppuration of the post-tracheal glands leading to an ul
opening into the trachea, was once observed by Steiner.

The appearances observed in other organs are for the m
the result of venous congestion. Œdema and congestion of th
is common in fatal cases. Meningitis of the base is a rare co
tion. The liver is congested, and hyperæmia and catarrh
stomach and intestines are also common. In the large intesti
catarrhal congestion may even give rise to dysenteric change
kidneys are also congested, and concretions of urates are ofte
in the straight tubules of the pyramids. General dropsy is a
sional complication. Amyloid changes in the liver, spleen, a
neys, which are sometimes present, must be regarded as pre-
and accidental conditions rather than as results of the pul
disease.

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[1] Loc. cit. i. p. 436.
[2] Legendre and Bailly only found five cases of tubercle in twenty-seven of
Pneumonia (loc. cit. 215). Steiner (loc. cit.) describes accumulations of nuclei
ring in the interstitial tissue of parts affected by collapse, proceeding to such a
as to fill the cavity of the alveoli. Other observers describe in the chronic
collapse a thickening of the interstitial tissue. I cannot but believe that such
lations of nuclear growth as Steiner describes are of a tubercular nature. Stein
of miliary granulations of tubercle in the pleura as not uncommon. I am
to believe, in spite of the recent statements of some high authorities, and par
of Niemeyer, that cheesy changes in the lung are a very rare event when not
or associated with tubercle.
[3] The patency of the foramen ovale with attendant cyanosis was observed, a
by Jörg, to be a frequent complication of collapse (Diss. de Pulmonum vitio
16), and also by Weber (Path. Anat. der Neugeborenen, ii. 39).

DIAGNOSIS.—The diagnosis of Broncho-pneumonia may be occasionally difficult, as may be inferred from the preceding description of its symptoms and physical signs. The chief points requiring to be alluded to here are the actual existence of Pneumonia in some cases of bronchitis, and its distinction from collapse in others.

Under all circumstances, the indications afforded by the thermometer are most valuable, and often aid in the interpretation of the physical signs.

The latter are often obscure ; but when sufficient lung-tissue is consolidated to alter the qualities of the percussion-resonance and of the respiratory murmurs, the following characteristics may aid in the diagnosis of Pneumonia from *collapse*, though, in the intermediate stages by which the latter passes into the former, many of them are but little available.

The loss of resonance is more absolute in Pneumonia, and the note, though sometimes tubular, seldom possesses any of the tympanitic quality occasionally observed in collapse.

The side is more retracted, and the sinking in of the ribs and elevation of the diaphragm are more distinct in collapse than in Pneumonia. Indeed, when the latter is extensive, falling in of the lower ribs is not observed.

In collapse the respiratory murmur is weak or *nil.* In Pneumonia it is bronchial or blowing, but seldom tubular, in the form of Broncho-pneumonia. Exceptions, however, occur on this point, and pure collapse may give a metallic tone to the respiration.[1] Râles are not heard as a rule in collapsed lung (though the possibility of their presence when dilated bronchi traverse the collapsed portion can hardly be disputed). In Broncho-pneumonia, râles more or less approaching the crepitant or sub-crepitant type are tolerably constant, though not invariably present. Vocal resonance and vocal frémitus are increased in Pneumonia, and the former may acquire a bronchophonic tone. They are both, as a rule, diminished over collapsed portions, without alteration of the quality of the vocal resonance.

Many of these points may, however, fail, and the distinction by the physical signs alone is not always easy. Under such circumstances, observations on the temperature materially assist the diagnosis.

Whether the condition of the patient be pyrexial or not, collapse alone will neither give nor increase fever ; and the presence of consolidation, together with the supervention or increase of fever, is at least an indication that some portions of this are due to true pneumonic changes.

Among other indications, the following, taken in conjunction with the pyrexia, are also of value in the recognition of the pulmonary disease :—The change in the characters of the cough from a paroxysmal and painless to a short, dry, and painful one ; the acceleration of the respiration and of the pulse, and the increased distress and rest-

[1] Ziemssen, loc. cit. 335.

lessness of the patient, may also serve to point attention to the true nature of the disease.

When Pneumonia occurs in the lobular form in the course of bronchitis or measles, without the intervention of collapse, the diagnosis may be much more difficult. In both diseases the increase of the pyrexia, which rarely attains in the simple bronchitis of children a temperature exceeding 100° Fahr.,[1] or its continuance in measles beyond the period of the specific fever, should, even in the absence of distinct pneumonic dulness, afford grounds for a strong suspicion of pulmonary mischief, and particularly when moist bronchitic râles are also present.

The distinction of Broncho-pneumonia from the lobar form of the acute primary disease may in measles be occasionally difficult when the patient is seen for the first time after a large tract of lung has been invaded. If the affection be purely unilateral, the difficulty is further increased. In most forms of Broncho-pneumonia the affection exists in both lungs simultaneously, though seldom to an equal degree.[2] In some cases, however, when collapse has preceded the Pneumonia, the peculiar pyramidal form of the dulness may aid the diagnosis. Moist râles in the opposite lung, or in other portions of that affected, are a further clue. The characters of the pyrexia in the two affections are in most cases a valuable guide, but it must be borne in mind that in the fever of the acute primary form a crisis may occasionally be wanting, and that its subsequent course also may be sometimes protracted.

The diagnosis from pleurisy, under these circumstances, rests on the same grounds as that of the acute primary disease.

The diagnosis of Lobular Pneumonia from acute tuberculization, or the recognition of tuberculosis as a complication of the Pneumonia, may at times be very difficult. This is especially the case when the disease originates in simple bronchitis; and when the general dissemination of moist râles, accompanied by pyrexia, may often closely resemble the phenomena observed in disseminated miliary tubercle. In measles and hooping-cough, a febrile state associated with pulmonary symptoms developed in the course of these affections would raise a presumption of its acutely pneumonic character.

The rapid development of signs of consolidation, with or without antecedent collapse, will, under all circumstances, favour the diagnosis of Pneumonia, but especially so in the two latter affections.

As points of minor value may also be noted the fixity of the râles in cases of tuberculosis, while the dyspnœa is often more distinctly disproportioned to the physical signs than it is in Pneumonia. Usually also the strength is less markedly or suddenly prostrated in tuberculosis than in Pneumonia.

[1] I have recently seen two cases in adults where a temperature of 105° was attained in the course of apparently uncomplicated bronchitis.

[2] Ziemssen and Krabler remark, however, that a double pneumonia may occasionally occur in measles so acutely as to be undistinguishable from the primary " croupous " form (loc. cit. 169).

Even in the later stages the difficulties may increase rather than diminish, since the progressive dilatation of the bronchi may closely simulate the formation of cavities from softening tubercle. The emaciation also and loss of strength in protracted cases of Broncho-pneumonia may bear a great resemblance to the phenomena observed in the later stages of tuberculization.

Under such circumstances the observer must often remain in doubt, though his opinion may be influenced by the previous health of the patients, by their constitutional or hereditary predisposition, and by the evidence or not of the existence of tubercle in the lymphatic glands.

THE PROGNOSIS of Broncho-pneumonia is of much greater gravity than that of the acute primary disease. Ziemssen records thirty-six deaths in ninety-eight cases in children, and in nine more various sequelæ remained. The mortality of Steffen's cases, also in children, amounted to forty-one out of seventy-two cases; in six of these, however, the lung affection was complicated by heart disease, noma of the external genitals, tubercle, pleurisy, and meningitis. Steiner records a mortality of one in three cases. The results of different authors vary as to the relative mortality of the disorder in the different diseases of which it is a complication;[1] but, as a rule, the acute forms appear to be less dangerous than those running a more protracted and chronic course.

The age of the patients affected has, however, a marked influence on the mortality. Bartels says that Broncho-pneumonia after measles was fatal in all the children who had not completed their first year. From ætat. one to five the mortality was 39 per cent., and after the age of five years it was 37 per cent. The collective mortality of all the cases enumerated by Ziemssen under one year old was as 1 : 1, occurring in equal proportions in measles, bronchitis, and hooping-cough:[2] of the whole number of Steffen's cases the mortality before two years of age amounted to 54 per cent.

The condition that most unfavourably influences the prognosis is the weakness of the patient, not only as affecting the direct possibility of recovery, but also as predisposing to further collapse. This statement explains the mortality at very early ages, and is specially applicable to hooping-cough, where collapse, which is itself due in great measure to exhaustion of the muscular powers, is frequently the direct agent in the production of pneumonic changes; and hence the

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[1] Ziemssen's data give the following percentage of mortality :—Measles, 25 per cent.; bronchitis and chronic bronchitis, 43 per cent.; and hooping-cough, 51 per cent. Steffen found the mortality from measles the greatest, amounting to five-sixths, but his cases of this disease were few in number, and affected children of very early ages. His mortality from the pneumonia of hooping-cough was as 8 : 10, and in bronchitis and chronic bronchitis, as 14 : 41 cases. Bartels' mortality in pneumonia from measles was 42 per cent., and he states that 80 per cent. of all the deaths in 573 cases of measles were due to the lungs.

[2] There appears to be some omission in Ziemssen's subsequent tables (p. 329), as the totals of those affected at different ages do not correspond to the whole numbers of his cases.

forms of Pneumonia occurring at late periods of the disease are not only indicative of loss of strength, but also predispose, by still further increasing the already existing weakness, to induce an extension of the pulmonary changes in which they originate.

For the same reasons a higher degree of fever, when of short duration, may be regarded as less unfavourable than a lower range of pyrexia, but protracted over a longer period. A temperature exceeding 105° Fahr. must, however, be considered as being of very serious augury. Among other symptoms which are to be regarded as unfavourable must be enumerated a great extension of the bronchitis over both lungs, signs of extensive collapse, increasing cyanosis, and diminished power of cough and expectoration, as shown by râles in the trachea and larger bronchi. To these must be added extreme frequency and particularly great feebleness of the pulse.[1] Somnolence and coma, indicative of mal-oxygenation of the blood, are also serious symptoms, not only in their direct indications, but also through the injurious influence produced on the respiratory muscles and on the heart by the impaired conditions of innervation of which they are the evidence. Convulsions in the later stages of the disease are a most unfavourable sign.

TREATMENT.—In the treatment of Broncho-pneumonia it must be constantly borne in mind that in the vast majority of cases this is a secondary disorder, and one whose very existence and mode of origin are very commonly indicative of weakness. This statement, which is applicable to all the forms of the disease originating in collapse of the lung, is hardly less true of the cases when, as in measles and diphtheria, the Pneumonia originates by direct extension of the inflammatory process from the bronchial mucous membrane.

When this fact is remembered it is scarcely necessary to mention all measures of treatment calculated to depress the powers of the patients, such as abstraction of blood by bleeding by leeches or by cupping, tartar emetic, calomel, and mercurials in general, except to state that their employment in such cases is only to be regarded with the strongest reprobation, since there is no doubt but that they tend to increase the mortality of the disease. Even when there is the temptation to abstract blood in order to relieve urgent dyspnœa, it must be remembered that the subsequent duration of the disease is long, that its tendency is to produce death by exhaustion, that all depletory measures diminish the muscular powers, and that by increasing the difficulty of expectoration they favour an increase of the collapse of the lung, which under such circumstances may speedily prove fatal.

In the earlier periods of the Broncho-pneumonia of measles, unless the fever is severe, an expectant treatment, with the administration of nutritious food and the employment of salines, is all that is absolutely

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[1] Barthez and Rilliet record a case where the pulse could not be felt during many days.

necessary. When, however, expectoration is difficult, and when the râles in the chest are abundant and dyspnœa is marked, the occasional employment of emetics is productive of considerable relief. Of these, ipecacuanha in emetic doses is the most serviceable, since its administration tends, simultaneously with the evacuation of the contents of the bronchi, to favour a freer and looser secretion, and thus to ward off the tendency to collapse. The beneficial effects of emetics are seen in the relief of dyspnœa and in the diminution of cyanosis, and the temporary depression which they sometimes occasion is speedily recovered from in consequence of the relief which they afford to the breathing. If the cough is very troublesome and frequent, opiates may be cautiously used, particularly as the continued expiratory efforts, when prolonged and forcible, are among the chief agencies by which collapse is produced. This statement, though applicable to all forms of the disease, is especially true of those originating in hooping-cough; and in this disease other agents capable of diminishing the violence of the spasmodic cough, such as opium, belladonna, alum, zinc, or the bromide of ammonium, may be also employed with advantage. In the administration of opiates, however, narcotism is to be carefully avoided; and it must be remembered that this effect is easily produced in all diseases in which the aëration of the blood is impeded. Such an effect is also doubly dangerous through the diminished muscular power which it entails, and the doses given must therefore be in the minimum amount sufficient to allay the cough.

In cases where the expectoration continues difficult and the pulse is weak, sedatives may with advantage be combined with the carbonate and muriate of ammonia,[1] together with small doses of the vinum ipecacuanhæ ($\mathfrak{m}$ iij. to $\mathfrak{m}$ v.), and with preparations of benzoic acid and of senega.

When prostration is more marked, wine or brandy should be given in doses proportioned to the age of the patient. For infants it is best to commence with brandy in doses of from 5 to 10 drops every two or three hours, gradually increasing both the quantity and the frequency of the dose, until a decided effect is produced on the pulse and on the respiratory movements. It may occasionally be necessary to give as much as ʒss, or even ʒj, every hour, to infants of a few months old, though it is very rarely that such an amount is required during long periods. Under its influence, however, both the pulse and the respiratory movements become slower, and the latter deeper and fuller; the convulsive movements are arrested; the prostration and semi-coma sometimes observed are diminished or disappear, and the pallor mingled with cyanosis gives place to a more natural colour. It is most important that the employment of these agents should not

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[1] The preparations of ammonia are best administered to infants in milk. Mr. Squire has recently, at my request, made a number of experiments in order to discover the best method of disguising the unpalatable flavour of the muriate of ammonia, and has done so very successfully by combining it with the tinct. limonis and sp. chloroformyl.

be too long delayed; and when dyspnœa and prostration are extreme, the action of emetics may often be assisted by their administration. When from the intensity of dyspnœa deglutition has become difficult, it is occasionally advisable to administer stimulants combined with beef-tea, or with Liebig's extract of meat, or with egg or milk, by the rectum, until the patient has rallied and the state of depression alleviated. Under these circumstances also, quinine and the preparations of bark may often with advantage be combined with the expectorant remedies. If diarrhœa exists, it must be carefully combated by astringents, and by bismuth combined with these and with small doses of opium.

Stimulating liniments or the application of mustard poultices to the chest are advisable in all these conditions, but the employment of blisters is highly undesirable, since they weaken the patient, and in children are liable to cause a dangerous sloughing of the subcutaneous cellular tissue.

The method of treatment by cold compresses applied to the chest has received the strongest encomiums both from Bartels and Ziemssen, and from their statements and observations it appears to be one of the most valuable of our remedial agents, particularly when the fever is high ; nor does any danger appear to arise during its employment from any extension of the pulmonary disease. It appears to operate favourably in two directions, both by increasing the strength and depth and by lessening the frequency of the respirations, and also by the reduction which it effects in the temperature,—a result which appears unattainable by any other agent, at least in an equal degree.[1]

Bartels particularly insists on the benefit derived from the first deep inspirations excited by the application of the cold compress to the thorax, in promoting the expansion of the lung, and warding off the threatened danger of increasing collapse.

The reduction of temperature which follows their application is also very remarkable. In eight hours Bartels witnessed a fall from 105·25 to 96·8° Fahr., or of more than 8° Fahr. ;[2] and Ziemssen, within seven and a half hours, observed in the rectum a fall of 5·8° Fahr. This effect is not, however, permanent, for after a few hours' intermission of the treatment the temperature again rises, and the application of cold requires therefore in some instances to be continued during some days before the temperature is permanently reduced to the normal standard.

This treatment needs to be carefully watched, since when it is prolonged without intermission, a dangerous degree of depression may ensue. It is, however, rarely observed until after several hours' application of cold ; but in a case recorded by Ziemssen it was noticed within half an hour after the treatment had commenced. The face

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[1] Digitalis has little or no effect on the temperature of Broncho-pneumonia, and only very rarely has it any influence in lessening the frequency of the pulse.

[2] This result was obtained in the axilla, and is therefore less trustworthy than Ziemssen's as regards the general effect on the temperature of the body.

under such circumstances becomes pallid, the eyes sunken, the skin cold, and the pulse small and almost imperceptible. This alarming state is said by Ziemssen speedily to disappear on the temporary intermission of the cold applications, and neither in five cases of this nature observed by him, nor in a similar one occurring in an infant aged only thirteen months, recorded by Bartels, did any further injurious or fatal consequences ensue, and the treatment was repeated with favourable results after the depression thus excited had passed off.

Although the first application of the cold compresses is often disagreeable to the patients, a remarkable improvement usually appears speedily in their general state. Both the pulse and the respirations fall in frequency, and the former becomes fuller and the latter deeper. The pulse may fall from 170 to 130, and the respirations from 80 to 34 in the minute. The appearances of cyanosis simultaneously diminish, and the patient, previously restless, often sinks into a sound sleep while still enveloped in the cold wet cloth. With the intermission of the applications the pulse and respiration again increase in frequency, simultaneously with the rise of temperature which is then observed.[1] It is probable, as Bartels has remarked, that these favourable effects are not attributable solely to the artificial abstraction of heat, but that they are also due in part to a diminished destruction of tissue throughout the body, and that thus the production of an excess of carbonic acid, for the elimination of which the diseased lungs are incompetent, is also favourably held in check.

The favourable effects of this system are strikingly shown by Bartels' results ; for whereas under other methods of treatment he lost, in the pneumonia following measles, seventeen out of twenty-six cases, or 65 per cent., the mortality after its adoption, and when no other remedies were employed, amounted to only thirteen out of forty-two cases, or little more than 30 per cent. In some cases, even when it was employed, the duration of the pneumonic consolidation was, however, very protracted, extending in one instance to eight weeks.

Under all the circumstances of the disease, the hygienic treatment of the patient requires to be carefully attended to. Fresh warm air, and the avoidance of draughts, are most important points to be insisted upon, and flannel should be worn next to the skin.

During convalescence the same precautions are to be observed, and the liability to relapse must be constantly remembered. It may be necessary during some months to enforce the extremest precautions against causes of catarrh, and during the winter months a confinement within the house, but in well-ventilated rooms, which should have a southern aspect, may be absolutely necessary in the case of children of delicate constitutions, or in those whose strength has been much reduced by the disease. Careful attention to diet, and the maintenance of the nutrition of the patient, are also most important. The admi-

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[1] Even in the most advanced and seemingly hopeless cases, and when the eyes appeared insensible to the stimulus of light, Bartels observed a gradual return of power, and a finally favourable result, after the adoption of this method.

nistration of cod-liver oil and of preparations of iron, and small quantities of wine sometimes given two or three times daily, are often necessary to complete restoration ; while in many instances a change of air, particularly in children brought up in large towns, is the most effective remedy that can be employed.

When the consolidation becomes more chronic, and is attended with profuse secretion from the bronchial mucous membranes, and particularly when dilatation of the bronchi exists, as shown by coarse râles in the chest, the same method of treatment must be sedulously followed. The administration of stimulants must, however, be pursued with caution when any tendency to pyrexia persists ; and under these circumstances, when the weakness of the patient appears to require their employment (a condition frequently observed), it is well to administer them during apyrexial periods of the day, which must be carefully ascertained and subsequently watched by the aid of the thermometer.

The use of inhalations, and particularly of turpentine, has under these circumstances been tried by Ziemssen, and with some favourable results.

OTHER FORMS OF SECONDARY PNEUMONIA.

Pneumonia occurring in the course of Bright's Disease may in some cases present no special variations from the characters observed in ordinary acute Pneumonia. In others it may begin, as before stated, in collapsed portions, resembling more or less in its course and characters the Lobular Pneumonia of children. Even when this is not the case, the characters of the primary disease are modified by this complication. The pyrexia is usually moderate, but the sputa tend to be thin and watery, and there is a considerable liability to œdema of the lungs and to consecutive grey hepatization. The tendency to pericarditis is also, I believe from my own experience, increased by this complication.

Rosenstein[1] has observed that when Pneumonia supervenes in Bright's disease, the total quantity of the urine is diminished, but that, in contradistinction to what is observed in other conditions, the amount of urea and the specific gravity still remain below the normal standard. M. Jaccoud has drawn attention to the fact that a low specific gravity with diminished water and a minor amount of urea may aid in the diagnosis between chronic Bright's disease complicated by Pneumonia, and the cases where albuminuria occurs as a complica-

[1] Path. Therap. Nieren. Krank.—The following are the results of Rosenstein's analyses :—

Day of Pneum.	Amount cc.	Density.	Nacl. grammes.	Urea. grammes.	Albumen. grammes.
1st	600	1013	2·4	3·6	1·8
2d	650	1013	2·47	4·37	3·25
3d	600	1012	2·10	4·5	2·4
4th	700	1012	2·24	5·85	2·8
5th	700	1012	2·8	—	2·1
6th	580	1012	2·32	4·98	1·7
7th	190	1013	0·76	1·04	0·95

Death on the seventh day from suppuration and œdema of the lung.

tion of the primary disease, but in which, nevertheless, an excess of urea is commonly present and the urine retains a high specific gravity.

The dangers of this complication have been already alluded to.

Rayer[1] remarks that Pneumonia occurring as a complication of diseases of the urinary organs, associated with alkaline urine, has the tendency to render this secretion acid, and his statement is confirmed by Grisolle.

The Pneumonia occurring in the course of the Acute Febrile Diseases has its features materially modified by the special symptoms of these, and presents in consequence so many variations that no general description will embrace the whole of the phenomena observed.

In *typhoid fever* it usually commences during the later stages. Its invasion is rarely marked by rigors, but commonly by a rise of temperature above the standard previously maintained. Fuller data are wanting on the subject of its further course. In cases which I have observed, the phenomena of crisis were absent, and when improvement has taken place it has been by a gradual fall of temperature, which may only occur after the pyrexia of the primary disease has subsided; the resolution of the infiltration also is often slow and protracted. Greatly increased prostration and asthenia attend this complication. The pulse and respiration are accelerated and their ratio is perverted, and the increase in the rapidity of breathing, together with that of the pyrexia, may be the first indication of the changes in the lung. Cough may be almost entirely wanting, and the rusty sputa are, as observed by Louis, comparatively rare. The insidious mode of invasion of Pneumonia in these cases renders a frequent examination of the chest necessary in all cases of continued fever. The Pneumonia commonly assumes the anatomical characters of red hepatization, but the tissue is softer and more gorged with blood than in the acute primary form. Various stages of transition to grey hepatization are also found.

The Pneumonia arising from *diseased heart* presents also in many cases the features which are most characteristic of catarrhal or broncho-Pneumonia. This is especially evident in cases of marked disease of the mitral valve. The congestion thus produced in the bases of the lungs may be so extreme as to give rise to dulness on percussion, but the respiratory murmur at this stage is blowing rather than tubular. There is almost always chronic cough, with frequent exacerbations, until finally a more acute attack supervenes, attended by œdema of the lungs, and accompanied by coarse râles. The sputa are bronchitic, clear, watery, or frothy, sometimes blood-stained, but rarely distinctly rusty or tenacious. The dulness gradually progresses, and the breathing becomes more bronchial in character; and these physical signs, accompanied by increased vocal fremitus and by intensified though rarely by bronchophonic vocal resonance, often appear in scattered patches, which may vary in site from day to day. Rigors are hardly ever observed as the pneumonic changes progress: the invasion is gradual, and the tem-

[1] Maladies des Reins, i. 573.

perature is often scarcely elevated even when the pneumonic consolidation is considerably advanced.

Portions of lung thus affected are found intensely œdematous, but airless; the section is smooth or indistinctly granular, and the pneumonic consolidation usually begins in patches of variable size, in which all gradations of the inflammatory changes may be observed. They finally, however, tend to coalesce and to pass into the condition of grey hepatization, yielding, from the œdema present, an excessive amount of turbid fluid on pressure.

The frequency with which Pneumonia occurs in the course of *other chronic and exhausting diseases* has been already referred to. It is usually of the hypostatic form, and tends to appear in scattered nodules in the midst of congested tissue; and, as before stated, it is not improbable that in many cases it is produced through the intermediate mechanism of collapse. The nodules are very soft and friable, often whitish, distinct, and break down easily into a pulpy débris.

Its invasion is rarely preceded by rigors; cough and sputa may be alike absent, and the only evidence of the disease until detected by the physical signs, are the supervention of pyrexia—commonly moderate in amount—and some acceleration of respiration. Pneumonia is, under these circumstances, very frequently the direct agency by which a fatal termination is induced.

The Pneumonia from *pyæmic conditions*, or Metastatic Pneumonia, has been already described in this work under the head of Pyæmia. (See Vol. I.)

The treatment of these forms of Secondary Pneumonia rests upon the same principles as have been described as applicable to all the adynamic forms of the acute primary disease. Briefly, it may be described as consisting almost exclusively in the administration of sufficient quantities of stimulants and support. In the Pneumonia of the continued fevers, these indications are especially called for, and considerable amounts of alcoholic fluids must sometimes be given in order to sustain the patient's strength.

In the Pneumonia complicating heart disease, digitalis may often be given with advantage, in moderate doses, when the pulse is rapid and small; but the administration of stimulants is by no means to be omitted. The carbonate and muriate of ammonia may also be used with benefit under these circumstances.

APPENDICES TO ARTICLE ON ACUTE PNEUMONIA.

APPENDIX A.

ON THE PULSE IN ACUTE PNEUMONIA.

THE accompanying woodcuts represent the chief forms assumed by the pulse in various stages and in different degrees of severity of the disease.

The first three were taken from a man, aged thirty, with consolidation of the lower two-thirds of the right lung, and they depict the gradual improvement following the crisis and during the administration of stimulants.

1.

2.
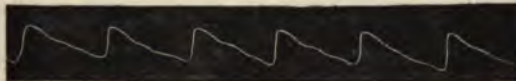
3.

4.
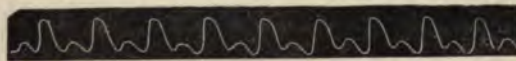
5.
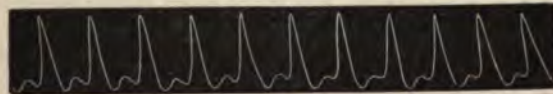
6.

No. 1 was taken on the eighth day, when the temperature was 104·8, and when great prostration was present. The frequency of the pulse was 110.

It was distinctly jerking and excessively compressible. The number o
respirations was forty.

The tracing shows a slight tendency to dichrotism. The recoil is
and the curve with the convexity downwards, corresponding to the n
condition of arterial tension, is almost entirely absent.

On the evening of that day the first marked remission took place by
to 102·8, and by the ensuing evening the temperature had fallen to
above which no further rise ensued.

No. 2 represents the pulse on the ninth day of the disease and aft
temperature had fallen to normal, and the patient had taken during t
four hours rather more than three ounces of brandy, in doses of ʒij.
two hours, with carbonate of ammonia gr. iij. every four hours.

The arterial tension and also the cardiac power are shown to be
increased, by the prolongation of the recoil, and by the even, gradual,
ward curve of the descending line. The tendency to dichrotism h
almost disappeared.

On the eleventh day, as seen in No. 3, the pulse had nearly regaine
normal standard. The same treatment had been persisted in througho
period, though the brandy was given at the longer intervals of fron
to four hours. During this period the pulse had fallen to 72 and the
rations to 30 in the minute, and on the following morning the
proportion of 84 to 20 was regained in their ratios.

No. 4 is a tracing taken on the fifteenth day of the disease, from a
dissipated habits, who was accustomed almost daily to drink ex
quantities both of beer and of spirits. The Pneumonia involved the
two-thirds of the right lung. The disease in this case ran a pro
course. An imperfect crisis took place on the ninth day, but the
returned, and only subsided on the twenty-second day, with occ
slight subsequent exacerbations occurring until the thirtieth ; on the fi
day, when this tracing was taken, the temperature was 100°, the pu
and the respirations 32. Puriform sputa, indicative of grey hepat
appeared early ; and constant delirium with intense prostration, and
perspirations, were prominent symptoms throughout the case.
quantities of brandy were necessary from the outset, and from the el
to the twentieth days (including therefore the time when this traci
taken) brandy was administered in doses of an ounce and a half ever
continuously. The pulse was very weak throughout, and was freq
intermittent. The tracing shows great deficiency in cardiac powe
arterial tension, but dichrotism is not observed here.

Tracings 5 and 6 are those taken on the sixth and seventh days in
ending fatally on the tenth day. The Pneumonia was double, a
nearly the whole of both lungs. Pericarditis was also present. Th
tracings show an extreme degree of dichrotism, and in No. 6 " hyper-
tism " (Anstie) is seen in the line of the recoil falling below the level
rest of the tracing. The temperature on these days was respectivel
and 104·8°. The pulse was 120, and the respirations 60 in the n
Brandy was given freely in this case, but not in the same amount as
last instance.

APPENDIX B.

ON THE RETENTION OF CHLORIDE OF SODIUM IN THE SYSTEM, AND ITS PRESENCE IN THE SPUTA IN PNEUMONIA.

A diminution of the quantity of chloride of sodium in the urine is common to a great number of febrile diseases. It is not, however, constant in them, nor is total suppression invariably observed in cases of Pneumonia. It would appear, therefore, that it is governed by some of the general laws of pyrexia; and although in Pneumonia the sputa and also the inflamed pulmonary tissue are found to present a considerable amount of chloride of sodium, as pointed out by Dr. Beale, yet this is hardly sufficient to account for the deficiency below the normal average (177 grains, Parkes). Dr. Beale found in one case that while the urine was absolutely deficient in chloride of sodium, the amount contained in the sputa was 10 per cent. of the solid matters. Dr. Beale thought that the chlorides were attracted to the inflamed lung as a consequence of the rapid cell-formation taking place there. When the chlorides were reappearing in excess in the urine, a similar excess was found in the serum of a blister, amounting to 8·93 of the solids, which Dr. Beale attributes to re-absorption from the lung during the period of resolution. (It must be remembered, however, that the serum of a blister is also an inflammatory product.) In a case ending fatally, the following proportions of chloride of sodium were found by Dr. Beale in different parts :—

Chloride of Sodium.	Per cent. of solids.
Urine	0
Blood from heart	0·68
Hepatized lung	2·59
Healthy lung	1·43

It would appear desirable that some analyses of the blood should be made during the period of absence of the chlorides from the urine.

I subjoin analyses of the urine and sputa in a young adult man, the subject of double Pneumonia, in whom, however, the temperature did not rise to any marked height. The analyses of the urine were conducted for me by my friend and then clinical assistant, Dr. Poore ; those of the sputa were conducted by Dr. Meusel, assistant to Prof. Williamson, in the Birkbeck Laboratory in University College. The case is so far complicated that until the tenth day the patient took daily 40 grains of hydrochlorate of ammonia, which would probably increase the amount of chlorides both in the urine and sputa. It will be seen, however, that in the early days the amount excreted in the sputa by no means complemented the deficient excretion by the urine. No cause could be assigned for the diminution of the urea on the eleventh day. A similar decline will also be noticed in the urinary chlorides from the eleventh to the fourteenth days. The patient perspired freely during this period, and possibly a considerable amount of chlorides may have been thus

eliminated by the skin, though the amount of the water of the urine
affected than the chlorides during this period.

Day of disease.	Temp. max.	Urine. Amount in c. c.	Sp. gr.	Urea. Grains.	Chlorides. Grains.	Sputa Chlorides. Grains. Total.	Total e Chlorides and S Gr
3rd day	102 8	..	..	..	..	} 17·6	..
4th ,,	103	..	..	..	..		..
5th ,,	101·2	970	1020	598·68	11·19	} 15·7	19·7
6th ,,	101·4	870	1020	535·72	16·786		24·5
7th ,,	101·5	1450	1019	792·71	50·204	5·37	55·5
8th ,,	100	1210	1020	670·82	64·142	6·0	70·7
9th ,,	99	1560	1020	792·79	95·896	6·34	102·2
10th ,,	100	1830	1016	605·86	119·658	3·803	123·4
11th ,,	99·8	1510	1015	488·33	36·806	2·78	39·5
12th ,,	99	1370	1015	506·35	71·619	..	..
14th ,,	98	1860	1015	657·20	48·756	..	..
15th ,,	97 (?)	1380	1013	382·33	147·955	..	..
16th ,,	98	2135	1012	509·58	50·880	..	..
17th ,,	98·5	1800	1011	388·26	157·850	..	..
18th ,,	98·5	2050	1010	473·5	115·808	..	..

The amount of urea subsequently varied between 385 and
during the succeeding five days. On admission the right base
affected, the left became implicated on the fourth day. Free p
commenced on the seventh day and continued on subsequent
sputa, which at first were copious, on the ninth day had lost their
and had become bronchitic in character; on the tenth day they
diminished in amount, but on the eleventh some rusty tinge rema
physical signs had only completely disappeared on the thirtieth d

APPENDIX C.

The cause of the granular appearance of a pneumonic lung
subject of much dispute. The question will be found discussed
the works of most writers on the subject of Pneumonia, part
Laennec, and also by Andral, " Clin. Médicale;" Chomel, " Lec.
Pneumonie;" Dr. Williams, art. "Pneumonia," Cycl. Pract. Med.
"Works," Syd. Soc. Ed.; Hodgkin, " Morb. Anat. of the Mucous
Membranes," vol. ii. It was by these writers, in varying degrees, at
swelling of the walls of the air-vesicles, to interstitial exudation in
and to the filling of the air-vesicles themselves with exudation
The merit of having first distinctly asserted in this country that
tion took place into the interior of the air-vesicles is claimed for Dr
and Dr. Hodgkin admitted that his views on the question had bee
by Dr. Addison. Dr. Addison's statements on this subject are
somewhat contradictory, as it would appear from his writing
regarded the solidification of the lung during the stage of red h
as due to the swelling of the walls of the air-vesicles (loc. cit.
and that even grey hepatization was attended with a similar chang
p. 22), and that at a later stage the softening of the walls admitt
albuminous material being poured into their cavities." In anoth

however, he states (loc. cit. p. 18) that "Pneumonia has its original and essential seat in the air-cells of the lungs, and that the ordinary pneumonic products are poured into these cells." Dr. Addison's editors and former pupils assert that he distinguished red hepatization from grey hepatization by the fact that the former consists in the swelling and gumming together of the walls of the air-cells without effusion into them, and that the latter consists of an albuminous effusion into the cells. (Editor's preface, loc. cit. p. 25.) This distinction cannot now be regarded as tenable; for in the first stages of Pneumonia the inflammatory products accumulate in the interior of the vesicles, and the walls are unaffected except by vascular hyperæmia. In the recognition of the intra-alveolar exudation, Addison was, however, according to Virchow (Ges. Abhand. p. 725), preceded by Lobstein (Arch. Méd. de Strasbourg, 1835, No. 1). Virchow (loc. cit.) states that the intensity of the granular appearance depends on the solidity of the exudation, and that it is less marked in the lungs of children, of old people, and also in dogs, because the exudation in them is commonly more fluid in its consistence. The granular appearance may, however, be distinct in the lungs of children, though on a finer scale than in adults. It may be still a question whether this appearance may not be in part due to the post-mortem coagulation of fibrinous and other materials, which during life are semi-fluid; and whether the exudation matter in the lungs may not undergo changes similar to those which ensue after death in other organs, such as the liver, the spleen, and the muscles (as shown by Kuhne's researches), through which they acquire increased firmness after life has ceased. Toulmouche (Gaz. Méd. x. 489) found in pneumonic lungs examined very shortly after death, that a quantity of fluid blood escaped from the cut surface.

APPENDIX D.

THE ORIGIN OF EXUDATION AND OF CELL-PRODUCTS IN INFLAMMATION.

The account of Cohnheim's researches may be found in Virchow's Archiv, vol. xl. 1867. It is due to earlier observers to state that although Cohnheim has by means of woorara found an admirable method of observing the escape of the white corpuscles through the walls of the blood-vessels, and has reduced it to a true demonstration, he was, however, anticipated in his observation by Dr. Addison of Brighton (Exp. and Pract. Researches on Inflammation, 1843; Healthy and Diseased Structure, 1849), by Dr. Waller in 1846 (Philosoph. Mag. vol. xxix. pp. 271—398), (I am indebted to Prof. Sharpey for this fact), by Zimmermann (Prager Vierteljahresch. 1852, vol. xxxv. p. 145), and still more recently by Dr. Lionel Beale (Microscop. Journ. xii. 1864). Dr. Beale describes the so-called white cells of the blood as multiplying *in loco* from the germinal matter of the nuclei of the capillaries, and he states that portions of this germinal matter pass through the capillary walls, and grow externally into cell-forms in the exudation. If I rightly interpret Dr. Beale's view expressed in other places on this point, he regards the solidified parts of the exudation as the "formed material" produced by "germinal matter,"—an opinion corresponding in some respects with Virchow's, that fibrinous exudations are the product of tissues in an excessive state of nutritive activity. Virchow, indeed, believes that in most instances the

connective tissue, from the close relation which it bears to the lymph structures, is the origin of fibrinous exudation (Gesch. Abband. p. 1 Buhl, however (Sitzungsbericht der Akad. der Wissensch. zu Münc 1863, vol. ii. p. 59), has argued that, when this exudation occurs on mu surfaces, the material so produced may be the result of transformations eff in the blood-plasma by the agency of epithelial structures. Virchow cit., and also in Archiv, vol. iv. p. 310, and in Spec. Path. Therap. vol. " Entzündung ") has pointed out that the exudative processes of inflamm have a close analogy to secretions, and that the fibrinous exudations a times more or less interchangeable with those in which a material resem mucin is formed ; and further, that all degrees of transition, in respect t qualities of the exudation, may be observed between catarrhal and "croup inflammations. These statements have a great interest and an impo bearing in the processes observed in Pneumonia, where these transition the nature of the exudation may be observed in its different stages. serve also to show that the boundary-line between the so-called croupou catarrhal forms, on which some recent authors have especially insisted, no means so sharply defined as is now sometimes believed ; while i latter, as noticed by MM. Herard and Cornil (Phthisie Pulmonaire, p. a true fibrinous exudation may be occasionally observed.

APPENDIX E.

ON THE TREATMENT OF PNEUMONIA BY VENESECTION.

As it is still at least theoretically maintained by some that the statis cases of Pneumonia treated by venesection show a superiority for thi cedure over other methods, it appears desirable to give a short ske the principal data which are accessible on the subject. All statistics o subject are more or less beset with fallacies, but the final conclusions drawn from them appear to me to be those which I have stated.

The arguments in favour of venesection rest chiefly on the data giv Louis[1] and Grisolle,[2] with whom also may be ranked Wunderlich,[3] wh recently supported the same view.

The weight of Louis and Grisolle's argument goes to show that case early, within the first four days, have a more speedy recovery than thos at later periods. Louis stated that cases bled within the first period h average duration of seventeen days, those within the second, an average tion of twenty days ; and in a second series he contrasts the duration the same circumstances, as being in the first instance between twelve and teen days, and in the second between fifteen and eighteen days. G states that in patients bled in the "first stage," convalescence began tenth day and was completed within three weeks ; while in those bled i second stage, convalescence began on an average on the twelfth day, and were discharged on an average on the twenty-second day. Both Lou Grisolle date convalescence from a day or two after the cessation

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[1] Rech. sur la Saignée.   Loc. cit.
[3] Arch. Phys. Heilk. 1856, xv.

fever. If, however, these data are compared with the periods at which it is shown that the fever naturally tends to decline without active inter- ference, it would appear not unjustifiable to infer that, regarded from the "positive" side, this evidence has no bearing on the absolute value of early bleeding, though demonstrating the relatively injurious effect of late bleeding in the disease.

Looking at the general results of these test cases, we find, however, that the mortality under Louis[1] amounted to 32 out of 107 cases, or 30·8 per cent., while that of Grisolle in 233 cases was 15·8 per cent., or 10 per cent. for the earlier bleedings, and 17·5 per cent. for the later; a mortality which in Louis' cases is vastly in excess of the average results of an expectant treat- ment, and in Grisolle's is only so to a less degree.

For an absolute comparison of the results of the bleeding and non-bleeding plan by the same individual, the most authentic data are those of Grisolle, Wunderlich, Huss, Dietl, and Dr. Todd. The two former have at- tempted to show that a number of cases treated by venesection have on the whole a shorter duration and a more favourable course than those in which no abstraction of blood is practised. Grisolle's data only rest on a comparison of eleven mild cases left to absolute expectancy (*including a rigorous French diète*), and thirteen of the same type treated by bleeding. In the former, he states that the convalescence only began on the tenth day, and the disappearance of the physical signs was protracted to the twenty- second or thirtieth, while in the latter the fever disappeared on the average on the seventh, and the physical signs on the twelfth days. These data have a certain incontestable value, but the number of cases is too small to weigh largely in the balance of evidence derived from the natural history of the disease.

Wunderlich's evidence is also in favour of bleeding. He gives a total of 190 cases, with an average mortality of 11·57 per cent.; 76 were treated without bleeding, with a mortality of 17·10 per cent., and 47 with bleeding, with a mortality of 6·38 per cent. The data as to sex, age, and complica- tions are, however, here also very imperfect.[2] In contradistinction, however, to the almost universal evidence of other authorities, he considers that out of eighteen cases of those bled on the first or second day, there was an almost immediate cessation of the pneumonic process in ten (*i.e.* crisis on the second, third, and fourth days),[3] and in five more a diminution of the pyrexia.

Traube (*Ueber Krisen und kritische Tagen*) had before asserted that as the natural tendency of Pneumonia is to a crisis in the uneven days,

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[1] Louis' own data afford one of the best evidences of the fallacies inherent in this class of statistics, for the percentage of mortality in his first series is greater in those bled during the first four days than in those bled later, in the proportions of 42·8 to 25 per cent.; a fallacy which Louis himself pointed out and rightly attributed to the com- paratively advanced ages of the patients who constituted the former class.

[2] Wunderlich gives collectively 114 cases in whom local bleeding (58) (?), general bleeding (47), and spontaneous hæmorrhage (9), occurred. The evidence deduced from the latter is, however, almost valueless; in seven of these epistaxis occurred simul- taneously with the crisis; in two females menstruation took place at an early period of the disease, and in one of these the crisis followed its cessation. Wunderlich gives no collective data, but only selects typical numbers; *e.g.* he only analyses thirty-two of the seventy-six cases where no bleeding was practised.

[3] This result is certainly remarkable when compared with the average frequency of the crisis on the third day. The amount of blood drawn was from seven to sixteen ounces.

active therapeutic interference by emetics or bleeding was likely to induce the crisis at these periods.

Thomas,[1] however, as the result of his researches made eight years later than Wunderlich's, but in the same hospital, asserts that bleeding has little or no influence in the reduction of temperature, and that the course of Pneumonia is identically the same, both under "active" and indifferent treatment; and the same result had been before arrived at by Von Baerensprung,[2] one of the earliest observers on this subject. Thomas further points out that the effect of bleeding shortly before the crisis is in some cases to produce an unnaturally low temperature after this has occurred. As such extreme depressions of temperature are almost always associated with marked prostration, this result of venesection can by no means be considered a desirable one.

Dietl's observations have been before alluded to, but they afford very strong arguments against the utility of venesection. His first observations[3] on 380 cases gave the following results :—

	Venesection.	Tartar emetic, large doses.	Expectant.
No. of Cases	68	106	189
Mortality per cent.	20	20·7	7·4

Dietl's statistics, in exact opposition to Grisolle's, show that cases left to a purely expectant treatment have a shorter duration of the pyrexial period, and a more rapid convalescence than those treated either by bleeding or tartar emetic, while the age of the patients and the complications present appear to have been nearly equivalent under all the systems tried. In a later publication[4] he gives the statistics of 750 cases (412 males and 338 females), all treated without venesection in the four years 1847-50, with a mortality of 9·2 per cent. Many of these were complicated (389 cases), including all the fatal cases; a large proportion (515 cases) suffered from "severe" dyspnœa. In the majority of those who recovered, the pyrexia lasted only from five to eight days, and the convalescence in most, from seven to fourteen days.

Huss's observations, extending over a period of sixteen years, brought him to the conclusion that during the time in which bleeding was indiscriminately practised in the hospitals of Stockholm, the mortality was greater than after it had been discontinued. During the former period of eight years, from 1840 to 1847, when venesection was generally employed, the mortality was 11·54 per cent., and during the succeeding eight years, from 1848 to 1855, when it was rarely resorted to, it was 10·21 per cent.; or the mortality of the former period exceeded that of the latter by 1·33 per cent. The average duration of the disease in those who recovered during the latter period was also shorter, being 20·9 days in the former, and 18·12 days in the latter.

The contrast of the effects of the two systems of treatment on the duration of the disease was markedly greater in the females than in the males;

[1] Arch. Phys. Heilk. 1864. It may be worth remarking that Thomas's data respecting critical days, before quoted, do not *include* these cases of Wunderlich's, though made in the same hospital. Thomas states that his observations date from 1860. Wunderlich's paper was published in 1856.

[2] Müller's Archiv, 1851, p. 174.

[3] Der Aderlass in der Lungen Entzündung.

[4] Wien Med. Woch. 1852. Schmidt's Jahresb. 1852, lxxvi. p. 30.

the average duration in the former being 7·6 days shorter in those not bled, while in the latter the difference was only 1·83 days.[1]

Huss asserts that there was no difference in the character of the pneumonias admitted during these periods, and he concludes that the difference is to be attributed solely to the influence of treatment.[2] Huss further adds

[1] An analysis of Huss's statistics shows that the corresponding numbers of the two sexes during these periods were as follow :—

1840 to 1847.		1848 to 1855.	
Males . . 773 cases.		Males . . 1,195 cases.	
Females . 147 ,,		Females . . 220 ,,	

[2] I have analysed Huss's tables, to see if any difference in age of the patients treated could have had any influence on these results, but the subjoined table, constructed from his, would appear to negative the possibility :—

AGE.	5—10.		10—20.		20—30.		30—40.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent of deaths.
1840 to 1847 Bleeding	3	33·3	104	7·6	430	6	321	10·5
1848 to 1855 Non-bleeding	6	0	125	4 8	611	5·2	495	10·8

AGE.	40—50.		50—60.		60—70.		70—80.	
	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.	No. of cases.	Per cent. of deaths.
1840 to 1847 Bleeding	125	22·4	49	18·3	7	14·2	1	0
1848 to 1855 Non-bleeding	238	16·7	76	23·7	22	27·2	3	66·6

The totals of those above and below æt. 40 may be represented thus :—

	UNDER 40 YEARS.		OVER 40 YEARS.	
	No. of cases.	Percentage of deaths.	No. of cases	Percentage of deaths.
1840 to 1847	858	9·5	182	20·8
1848 to 1855	1,201	7·4	341	19·3

It will thus be seen that a minor degree of mortality exists both for cases below and above 40 in the second half of this period of sixteen years, though the number of cases in both instances is larger than in the preceding period, but that in the later decennial epochs of life, after ætat. 50, the mortality, as shown by the first table (in the second

that, in his opinion, treatment by bleeding disturbs the natural tendency of the disease to crisis, a result before pointed out by Baglivi and adopted by Grisolle.

If we look at the effect of large bleedings indiscriminately practised, we see an enormous excess of mortality attending the treatment. Many of the data of these are very contradictory, but some are all but conclusive.[1]

Andral's mortality amounted to more than half his cases, or thirty-six out of sixty-five. Of the uncomplicated cases, nine were bled in the first stage of congestion, and two died; of thirteen bled in the second stage, five died; of seven bled in the third stage, *all* died; and of thirty-six complicated cases, twenty-two died.[2] The mortality of Bouillaud, according to his own report, was only 11 per cent., according to Peliatan's 12 per cent. Louis' mortality we have already seen. That of Chomel, according to Louis' report, was 32 per cent. Rasori's—a treatment complicated with enormous doses of tartar emetic—gave a mortality of 22 per cent. The mortality of Broussais is given at 68 per cent.

Many will contrast with these the results of Dr. Bennett before alluded to, which, even if considered exceptionally favourable, demonstrate that during a period extending over sixteen years a very large number of cases of Pneumonia, taken indiscriminately, *may* recover perfectly without venesection,[3] in the absence of serious complications, although presenting in some instances the most marked forms of dyspnœa and lividity of face, associated with double Pneumonia, or an extensive affection of one side involving in fifteen cases the whole of one lung. They further show that the period of convalescence and the duration of the disease do not exceed, and in many cases fall very short of, those observed when venesection is practised. Nor can we refuse to admit the conclusive evidence of his facts adduced from the same field of observation, that while the mortality from Pneumonia in the Royal Infirmary of Edinburgh, prior to 1848, and when large bleedings were practised, was 36 per cent., this diminished during eight years when bleeding was less employed to 21 per cent., and in the following nine years to 11 per cent., while in Dr. Bennett's own practice it has only amounted to 3 per cent.

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period of non-bleeding), absolutely *appears* to be greater. This is, however, probably fallacious, owing to the smaller number of cases on which they are calculated, since, in the face of the positive evidence to the contrary, it would be absurd to believe that venesection is relatively less dangerous at advanced ages. The data in Huss's tables give no means of forming further accurate comparisons on the influence of sex as compared with age, or of the complications present. The former, as far as I can gather from his tables, appears to be immaterial—the latter remains unanswered; but in dealing with such large numbers the probable influence of this cause of fallacy in the comparison is reduced to a minimum. The fluctuations in the mortality in different years appear, as already pointed out, to have been almost as great in the latter as in the former period.

[1] How uncertain such data may be appears from a communication made by Skoda to Dr. Balfour, that in 1840 he treated sixty-four females by large bleedings and tartar emetic, with only *one* death, but that in the same year the deaths among the males brought this average mortality to 12·5 per cent. (Brit. and For. Med.-Chir. Rev. 1846, xxii. p. 590.)

[2] Analysis by Dr. Markham.

[3] Nine of Dr. Bennett's cases were bled before he saw them, and to an extent varying from twelve to thirty ounces; sixteen more were subjected to limited bleeding by leeches or cupping: the amount so lost is calculated by him as varying from one and a half to eight ounces. These cases had not a more favourable course than those not so treated.

Dr. Todd[1] also pointed out that while the mortality from Pneumonia treated by bleeding combined with the use of tartar emetic amounted to one in six cases, this under a treatment by salines, nourishment, and support was only one in nine.

The argument that Pneumonia has changed its type and has acquired of late years a more asthenic character than it formerly possessed, is one on which exact data are necessarily wanting.[2]

Cullen's description of high fever and of a full bounding pulse applies to pleurisy as well as to Pneumonia, and instances of this class of symptoms in both diseases in young adults are not now, I believe, so very rare as they are sometimes stated to be. Such cases of Pneumonia, however, are those most likely to recover under any circumstances, and the statement that they "bear" bleeding better than the more adynamic forms of the disease is no proof of the utility of the treatment, but only of the minor degree of danger attending it under these circumstances, while there is abundant evidence that in the majority of such cases it is, to say the least, superfluous. Further, the soft and yielding pulse, which is the most common in Pneumonia, has been shown by Dr. Balfour, from Dr. Gregory's own records, to have been prevalent in his time, as now, but that it certainly formed no obstacle to his course of venesection. The argument of Drs. Balfour and Markham, that this asserted change of type was not recognised by some of the most acute observers then practising for nearly twenty years[3] after it was said to have begun with the epidemics of cholera and influenza in 1830 and 1832, and that bleeding was only discontinued after the experiment of an expectant treatment had proved its inutility, appears also a very forcible one. Bleeding was instituted and practised on the theoretical ground of humoralism, or on the mechanical (or "hydraulic," Balfour) ground of relieving the congested lung. It was held to be the almost universal remedy for fever and inflammation, irrespective of age or sex, and that at a period antecedent to the more perfect recognition of Pneumonia by means of physical diagnosis, which has been supposed to have extended the practice; but the final proof of experiment necessary to an inductive science was not applied by its advocates, and when thus applied the inutility of the treatment was immediately demonstrated. The analogy also of a change of type in fevers is a most doubtful one, since there is the strongest reason to believe that those referred to by Sydenham and others were not different manifestations of one disease, but were in reality the different forms of typhus, typhoid, and relapsing fever, whose

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[1] Clinical Lectures, p. 310.

[2] It is impossible to give more than a very superficial sketch of the able and elaborate arguments which have been advanced in this controversy. They will be found for the most part contained in the "Edinburgh Medical Journal" for the years 1856—1859, in papers by Drs. Alison and Christison, Sir Thomas Watson, Dr. Bennett, Dr. Gairdner, Dr. Balfour, and Dr. Mitchell. An admirable summary of them is contained in an article attributed by Dr. Bennett to Dr. Sibson, "The Bloodletting Controversy," in Brit. and For. Med.-Chir. Rev. 1858. The question of the theory of the "change of type" in acute inflammation is fully considered and negatived by Dr. Markham in the Gulstonian Lectures for 1864, "Bleeding and Change of Type in Diseases," and also by Dr. Balfour, "Hæmatophobia, an historical sketch," Edin. Med. Journ. 1858. To the latter the author is indebted for much of the earlier history of the schools of opinion on this subject. To Dr. Bennett's work on Pneumonia, and to Dr. Sibson's article, the author is indebted for many valuable statistical contributions.

[3] Dr. Balfour cites Dr. Alison as writing in 1844 ("Pathology"), that bleeding was the most important remedy for Pneumonia.

specifically diverse nature was not recognised until the writings of Sir W. Jenner. Dr. Balfour's historical researches have proved that this question is by no means a new one, but that it has descended to us from the followers of Pythagoras as opposed to those of Galen, and that even in the last century the same argument was advanced when the opponents of venesection had demonstrated its inutility in acute disease : Dr. Markham has also shown that it was supported by no less an authority than John Hunter. The opinion that such a change of type has taken place within more recent periods is further controverted by contemporary though indirect evidence. Laennec stated that the success of Dumangier in the treatment of Pneumonia without bleeding was equal to that of Corvisart, who bled freely ; and Dr. Balfour observes that at the very time Dr. Gregory was practising his enormous bleedings, Laennec asserted that the treatment of Pneumonia by tartar emetic alone had reduced its mortality to 3 per cent. The argument also involves this remarkable paradox, that a disease in its asthenic form is, in the abstract, vastly less dangerous than when presenting a sthenic type ; a paradox utterly confuted by our daily experience, not only of this, but of all other inflammatory diseases. This paradox appears in some of the ablest arguments advanced in support of the theory of a change of type in acute disease, since one of its most eminent advocates hails with satisfaction some signs of a return of the sthenic character.

The history of the origin of the change of treatment from venesection to a milder system also militates strongly against this view. Skoda and Dietl commenced their investigations on the results of expectant treatment on purely experimental grounds, and the former to the present day denies[1] any recognisable change of type in the forms of Pneumonia observed by him.[2]

As a final conclusion of the argument, it must, the author believes, be admitted on the evidence brought forward, that at no period since A.D. 1700 has blood-letting in Pneumonia been shown to be a general necessity in the disease ; and that although on more than one occasion since this date a change in the vital characteristics of the disease has been asserted, in order to explain the recovery of patients suffering from it, on whom no venesection was practised, yet that no valid proof has been afforded that such a change has really at any time taken place.

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[1] Allg. Wien Med. Zeit. viii. 1863. Schmidt's Jahrb. cxx. 34.

[2] A denial also maintained by Bouillaud, who is stated by Dr. Bennett to pursue his system of venesection *coup sur coup* with unabated energy, and with the fullest belief in its success ; while Grisolle, on the other hand, though still holding venesection to be the best treatment, asserts his belief that "la constitution médicale est moins inflammatoire qu'il y a vingt ans."

INTERLOBULAR PNEUMONIA.—INFLAMMATION OF THE INTERLOBULAR TISSUE OF THE LUNG.

THIS is the acute form, and in the human subject is a disease of the extremest rarity. Dr. Hodgkin [1] alludes to it, and it has been figured by Sir R. Carswell.[2] Dr. Stokes [3] also describes a case where " the substance of the lower lobe was completely dissected from its pleura by the suppurative inflammation of the subserous mucous membrane. This process also was found to have invaded extensively the inter-lobular and intervesicular cellular tissue, so as to cause this part of the lung to resemble nearly the structure of a bunch of grapes. All these nearly isolated lobules were surrounded by puriform matter, in which they hung from their bronchial pedicles." The exact condition of the vesicular texture is not described by Dr. Stokes, but his description would lead to the inference that it was in a state of hepatization. Rokitansky [4] has also described the disease in a form very similar to that met with by Dr. Stokes.

In Dr. Stokes' case death took place on the twelfth day from the first symptoms of the disease. Large râles were heard over the site of the change, and the characters of the respiration led Dr. Stokes to suspect the existence of a cavity. Renewed rigors and copious sweating occurred on the seventh day, and were repeated up to the time of the patient's death.

I have seen one instance of this change in the interlobular tissue, caused by the direct extension of a post-pharyngeal abscess along the posterior mediastinum to the roots of the bronchi. There was effusion with recent lymph in both pleuræ. The interlobular septa of the lower lobe of one lung were greatly thickened and of a yellowish colour, and were found to be the seat of a purulent infiltration. The lung tissue intervening between them was condensed, but was otherwise healthy, with the exception of several pyæmic abscesses scattered through its tissue. Thrombi were, however, found in several branches of the pulmonary artery. In this case also there were considerable pyrexia and frequent rigors followed by sweating. Dulness on per-cussion existed at the base for nearly a fortnight, and was attended by weak bronchial breathing and by fine crepitation, mingled with

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[1] Mucous and Serous Membranes, ii. 149.
[2] Museum Univ. Coll. c.b. 573. In his manuscript account of this drawing, Sir R. Carswell states that the patient was a man aged 60, who died of disease of the bladder without pulmonary symptoms.
[3] Diseases of Chest, 144.　　　　　　　　　　　[4] Anat. Path. 1861, iii. 72.

fremitus in these situations. The physical signs present, however, cannot be referred in this case exclusively to the condition of the interlobular septa, since other complications were present.

It may be noticed as worthy of remark, that this implication of the interlobular septa, though so rare in man, is the ordinary appearance of the pleuro-pneumonia of the bovine species. A full description of its characteristics has been given by Professor F. Weber of Kiel.[1]

There is no evidence at present existing that such a condition precedes those thickenings of the interlobular septa which are occasionally observed to follow inflammation of the pleura, but it is by no means improbable that the occurrence of this process in a modified form may be the origin of such appearances to which further allusion will be made.

[1] Virchow's Archiv, vi.

CHRONIC PNEUMONIA.

By Wilson Fox, M.D., F.R.C.P.

Synonyms.—Cirrhosis (?)[1]; Interstitial Pneumonia;[2] Lungen-Induration (Heschl), *German*; Sclerosis of Lung (Jaccoud); Fibroid Phthisis; Phthisie avec Melanose (Bayle); Scirrhus of Lung (Avenbrugger and older writers).

Definition.—A chronic induration of the pulmonary tissue, depending on a thickening of the walls of the alveoli by a fibrous growth, which causes a gradual obliteration of the cavity of the air-vesicles. This condition leads finally to contraction of the lung. It is commonly unilateral; it is frequently associated with dilatation of the bronchi; and it tends, either through ulcerations proceeding from these, or from secondary inflammation of the indurated tissue, to give rise to cavities in, or gangrene of the lung. It is associated with dyspnœa, with

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[1] Dr. Walshe, for whose opinion I entertain the most profound respect, and to whom as a former teacher I cannot sufficiently express my obligations, regards Chronic Pneumonia and Cirrhosis as independent diseases. The habits of inquiry which he taught his pupils will, I trust, serve as an excuse for one of them expressing an opinion on this point which differs in some respects from his own. The illustrations of the final effects of a pneumonia which has lapsed into a chronic state, appear to me to show that the result of the changes thus induced differs in no essential particulars from those which are met with in "cirrhosis" of the lung, in regard both to the induration of the pulmonary tissue and the dilatations of the bronchi, which so commonly are found in this state. M. Charcot is indeed disposed to make the existence of such dilatations a ground of distinction between the two diseases, but there is evidence enough to show that such dilatations are found in cases where induration has succeeded to an attack of Acute Pneumonia. They are not indeed so evident in the early as in the later stages of such cases, and the induration found in the latter is only a progressive change; but it appears to be an inevitable consequence of the disease if sufficiently protracted. The question is in one sense a purely pathological one, but as far as clinical diagnosis rests on a pathological basis it is not without its significance. There is abundant proof that thickening of the walls of the air-vesicles, resulting in the complete obliteration of their cavities, is a final result of Chronic Pneumonia, and it is this condition which is described in all (the few) authentic cases of "cirrhosis." I have discussed at some length the possibility of its origin in idiopathic changes independently of such inflammatory action. In the light in which I regard this state, and with this explanation, I have ventured to use Dr. Walshe's recorded case of this disease, which is the most perfect extant, and also his no less admirable commentary, as an illustration of chronic pulmonary induration.

[2] The term Interstitial Pneumonia also appears to me etymologically to express only very imperfectly the real character of this affection. The most important secondary effect of chronic inflammatory action on the tissue of the lungs is the thickening of the walls of the alveoli, and not of the interstitial tissue. It is indeed a question how far the latter is implicated, at least primarily, in this process.

cough, occasionally with fœtid expectoration, and with hæmoptysis. The course of the disease is protracted, but it tends to a fatal issue after considerable periods, through impairment of sanguification, dropsy, diarrhœa, and gradual marasmus, or through acute intercurrent diseases affecting the opposite lung.

HISTORY.—The condition of lung included under this title is one whose nature and pathological relations are as yet only imperfectly defined.

The views expressed by some recent pathologists respecting the inflammatory nature of the changes in the lung in many instances of phthisis would, if correct, necessarily involve the inclusion under this title of a very large proportion of cases hitherto regarded as tubercular, and indeed the estimate of the frequency of Chronic Pneumonia formed by different authors has varied largely with their opinions respecting the nature of tubercular changes. This division of opinion dates at least from the period of modern pathological research. By some authorities, and in particular by Broussais,[1] Cruveilhier,[2] Reinhardt,[3] and more recently by Lebert,[4] all tubercular changes have been regarded as essentially inflammatory in their nature.

Others, with Andral,[5] who recognised only the softer and more opaque granulations as tubercular, have regarded the grey granulation of Bayle, which many now consider the type of "true tubercle," as the result of a Chronic Vesicular or Lobular Pneumonia. A third series of observers—among whom may be named Gendrin,[6] the late Dr. Addison,[7] and, more recently, Niemeyer[8] and Colberg[9]—maintain

[1] See especially Examen, vol. i. Aph. 161 to 171; Hist. des Phleg. i. Proleg. p. liv. et vi. ib. p. 3; Examen, iv. 245, 402; Hist. des Phleg. ii. 385. Broussais recognised a pulmonary non-tubercular phthisis, but he regarded tubercles as the result of inflammation or irritation of the lymphatic tissues.　　　[2] Anat. Path. Gén. vol. iv. 1862.

[3] Annalen der Charité, vol. i.

[4] Gaz. Méd. de Par. 1867. Sur la Pneumonie disséminée chronique.

[5] Prec. Anat. Path. ii. 518, et seq.; Empis, De la Granulie. See also Reynaud, Mal. des Bronches, Dict. de Méd., vol. vi.　　　[6] Hist. Anat. des Infl. ii. 324.

[7] Works, Syd. Soc. Ed. Dr. Addison's statements on this subject are somewhat conflicting, and some passages in his writings would almost lead to the conviction that he held tubercle to be an inflammatory product; e.g. loc. cit. p. 33 : "Unless the simple transparent tubercle already alluded to can be considered as a separate and distinct body, there is not one of the varied morbid conditions coming under the denomination of tubercle which has not appeared to result from changes in or on the natural tissue. . . . These morbid changes have appeared to me perfectly identical with those of inflammation." "The immediate morbid changes produced by ordinary pneumonia and by phthisical disease are the same, with the exception of the albumen, ... being much more susceptible of organization, and consequently more likely to become permanent in the former than in the latter" (ib. p. 34). "If called upon to give an expressive name to tubercular phthisis, I should venture to designate the disease Scrofulous Pneumonia." In other places (e.g. p. 30), however, he treats of the grey granulation as occurring independently of inflammatory change; and at p. 49 he states, "However analogous and closely allied the abnormal condition which produces tubercle may be to that which constitutes inflammation, we cannot in the present state of our knowledge admit their identity." In another passage, however, he distinguishes two kinds of tubercle, a firm transparent, and a soft opaque form (loc. cit. pp. 49, 50).

[8] Lehrb. Spec. Path. Therap. Ed. 1868, ii. 233-5. Klinische Vorträge über die Lungen Schwindsucht, passim.　　　[9] Deutsch Arch. Klin. Med. ii.

an opinion precisely the reverse of Andral's, and assert that the greater part of the softer "tubercles," and nearly all caseous changes found in the lung, are due to a pneumonia which some of these authors have termed "cheesy" or "scrofulous." This view has also been in part supported by Virchow,[1] but it has been generalised by some recent writers to a wider degree than has been done by him.

It is undesirable in this place to enter further into the discussion of these widely diverse views.

They have however largely influenced, and particularly of late, the opinions expressed respecting some forms of induration of the lung classed under the head of Chronic Pneumonia, and even the descriptions given of this condition, and they appear to have caused not a little discrepancy of statement respecting its relative frequency.

Thus authors who, like Hasse, Grisolle, and Chomel, maintain the doctrines of Laennec respecting tubercle, assert that Simple Chronic Pneumonia is a disease of extreme rarity, and that it is hardly ever met with except when complicated with tubercles.[2] Grisolle[3] states that he has only met with six cases in twenty-five years, and only four where the acute disease passed into a chronic state; and Chomel[4] writes that in sixteen years, during which he performed nearly three thousand post-mortem examinations, he only met with two examples. Andral,[5] however, regarding the subject from a different pathological point, stated that he had met with the disease much more frequently than Chomel. Dr. Stokes[6] says that in his experience Chronic Pneumonia is a very rare affection, but that it is "difficult to define the meaning of the words Chronic Pneumonia, or to draw the line of distinction between it and that low irritation of the lung which is followed by tubercular infiltration." In the succeeding pages the author proposes to treat only of such forms of chronic induration of the lung as may be reasonably presumed to have been caused by processes in which tubercular changes have had no share. In this sense the disease is of great rarity, and examples of it can only be found in isolated cases scattered in different journals and in monographs on diseases of the lungs. The author's own experience would almost confirm the statement of Hasse, that it seldom occurs except in the presence of tubercles; for out of five apparent examples of the disease which have come under his own observation, in one only were the lungs found on microscopic observation to be free from tubercles. In the analysis of cases by other authors those cases will be spoken of as tubercular which present granulations—grey, or soft, or cheesy—in the lungs or other organs.[7]

[1] Wien. Med. Woch. 1856. Die Krankhaften Geschwülste, vol. ii. pp. 600 et seq.

[2] Hasse, Path. Anat., Syd. Soc. Ed., p. 225. This is admitted to a great extent by Prof. Niemeyer, but he explains the concurrence of cheesy products with tubercles by the theory that the tubercles are secondary to Pneumonia.

[3] Loc. cit. pp. 82, 338.   [4] Dict. de Méd. xvii. 223.
[5] Clin. Méd. iii. 491.   [6] Loc. cit. 353.
[7] The author feels considerable diffidence in thus somewhat dogmatically criticising cases by other observers, and he is aware that exception may be taken to the view here expressed,

which differs from the opinions entertained by many advanced path
day, but which has only been arrived at by him after a prolonged and
of this subject. The question of the nature of tubercle underlies the
and he can only shortly state here the opinion which he entertains, tha
is not only liable to "cheesy" degeneration, but that it is also capabl
or less permanent tissue by fibrous transformation; and the last-nam
his opinion, a much more important element in the history of tube
recognised. Also, that it consists of a multiplication of nuclei and
the interstices of which are occupied by a delicate fibre network or b
substance; that this growth may be peri-bronchial and peri-vasc
appears in the walls of the air-vesicles; that when found in the latte
but not always, accompanied by a proliferation of epithelial cells of
racter in the interior of the air-vesicles; and that in a large propor
"catarrhal," "gelatinous," and "scrofulous" pneumonias the cheesy
lung are accompanied by this "tubercular" infiltration of the wall
these "cheesy" changes may occasionally be due to fatty metamorpho
attended by destruction of the pulmonary tissue, but that in a far
cases they are due to a true tubercular change, and that even wl
direct cause of such changes in isolated spots, tubercles are almost
other parts of the same lung, and also in other parts of the system.
present this peculiarity of "cheesy" or "scrofulous" change, are th
ably those who are at the same time the subject of tubercle; and th
he is correct in stating that in the vast majority of cases such "ch
under the influence of the tubercular diathesis, and are mostly a
caused by the presence of tubercle. On the other hand, he is fully pr
Dr. Addison and Cruveilhier that a large proportion of the alteration
patients are due to attendant Pneumonia. This Pneumonia is commor
not destructive, it leads to a thickening of the walls of the air-vesic
fibrous tissue. This thickening takes place by means of a fibro-plast
gated and fusiform cells, independently of the tubercular masses befo
cular masses may, however, be mixed with these, and the two set
on *pari passu*, while the tubercular growths may either soften and breal
selves at later periods undergo the same fibrous transformation. Fi
of the lung tissue is therefore an exceedingly common event in tul
forms in fact, in one sense, a mode of cure of tubercle, as has been lo
mode of evolution of most forms of tubercular growth in the lungs is
to an inflammatory change, but it presents in addition other phenome
narily met with in inflammatory processes; and until the *purely* inf
tubercle is more distinctly proved than has yet been done, it appears
clinical sense, to maintain the separation of these processes. A disct
is, however, impossible here. As regards the co-existence of "chee

origin, though in not a few instances it can also be referred to past inflammatory conditions. To these, perhaps, may be added the induration of lung occurring in connection with heart-disease, and designated by Virchow [1] as the brown or pigmentary induration of the lung, syphilitic disease of the lung, and also certain rare conditions associated with non-tubercular ulceration.

ETIOLOGY.—I have already stated, in the section devoted to the clinical history of the acute disease, that I have only known one case of Pneumonia where the patient left the hospital without a perfect resolution of the physical signs in the lung; but I have also given instances where this process was protracted.[2] I do not think that cases of the latter class, in which a somewhat tardy but progressive improvement takes place, can properly be called instances of Chronic Pneumonia. Huss, however, dates the tendency to pass into the chronic state from the fourteenth to the twenty-first day of the acute disease. He says that this protracted course is somewhat more common in Pneumonia of the upper lobes, and that the Pneumonia of drunkards has a similar tendency. Grisolle states that Libermann has asserted it to be common amongst opium-smokers in China, and Dr. Stokes considers that Chronic Pneumonia ending in induration of the lung is more common after the typhoid forms of the disease. Chomel attributed to excessive bleeding an injurious influence in protracting resolution. The Pneumonia of the aged has also a similar tendency, particularly after the stage of grey hepatization has been attained. Circumstances interfering with convalescence, and fresh exposure leading to relapses, may also protract the course of the disease and give it a chronic character. Thus Broussais [3] gives three cases of induration of the lung from military hospitals, ending fatally on the twentieth, fifty-first, and ninety-first days after an attack of Pneumonia. In two of these, a condition of induration alone is mentioned, but in the second, the state described approaches closely to Andral's description of the red induration. Grisolle also states that the appearance of the lung, in cases of Pneumonia ending fatally within five or six weeks, presents but little difference from the characters of the acute stage,[4] though exhibiting a more marked degree of induration; the surface on section being somewhat smooth, but in other cases still presenting the granular character of the primary disease. The only case which I have met with

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[1] Archiv für Path. Anat. i. 463. This state is also alluded to by Andral (Prec. Path. Anat. ii. 517) ; Hasse (loc. cit. p. 227).

[2] Also Andral (Clin. Méd. iii. 550). A case where the signs of consolidation only disappeared at the end of four months.

[3] Hist. des Phlegm. i. pp. 13 et seq.

[4] Cf. a case by Bayle (Phthisie Pulmonaire, obs. 46, p. 373,—Pneumonia of three months' standing—red, firm hepatization ; also a case by Durand-Fardel (Mal. des Vieillards, p. 589), where death took place after two months, and red hepatization was found passing in spots into grey ; also (Ib. p. 594) a case of three months' standing, where grey induration existed at the bases, together with recent grey infiltration of one apex. Hourmann and Dechambre (loc. cit.) also speak of this protracted course as common. See also a case by Rayer (Gaz. Méd. 1846, p. 983), duration not stated ; also a case by Grisolle (loc. cit. p. 72), of a patient dying on the sixtieth day, when transitions from red and grey hepatization to firmer degrees of induration were found.

of this nature was in a man, aged forty-six, the subject of chronic albu-
minuria : cough, with hæmoptysis, began two months before admission,
but he was only compelled to leave off work a fortnight before ad-
mission into hospital. Dropsy in the legs had been present for six
months. The sputa were thick, puriform, and uniformly blood-stained.
The patient died suddenly three days after admission. The bronchi of
both lungs were dilated. Both apices presented a grey infiltration,
which was most marked in the left upper lobe, which was also con-
siderably indurated ; the kidneys were granular.

The condition of lung, however, most commonly described as
Chronic Pneumonia, is that in which the pulmonary tissue has under-
gone a fibrous induration, more or less deeply pigmented, usually
attended with complete obliteration of its vesicular structure, and
commonly, but not constantly, traversed by dilated bronchi. It is
this state which received from Sir D. Corrigan the name of "Cir-
rhosis,"[1] and which some modern English pathologists have regarded
as the result of an idiopathic change, which has also been termed
"Fibroid Degeneration of the Lung,"[2] or "Fibroid Phthisis."[3] The
condition has, indeed, been long known. It was described by Mor-
gagni,[4] and later by Avenbrugger,[5] under the title of "Scirrhus" of the
Lung, and by Bayle as "Phthisie avec Melanose," and the last-named
author recognised its occurrence independently of or complicated by
tubercular disease :[6] the same condition was also described by Laennec[7]
as occasionally complicating dilatation of the bronchi, and as existing
around tubercular excavations. The pigmented form was, however,
included by him under the term melanosis, which he regarded as an
independent disease, but which Andral first showed to result from
a chronic inflammatory action.

The difficulty in arriving at a conclusion respecting the mode of
origin of this state is, however, very considerable, owing to the length
of time during which pulmonary symptoms may exist before death,
and also in many cases from the incompleteness of the reports furnished.
I have, however, analysed thirty-nine cases[8] returned as "Chronic

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[1] Dublin Journ. 1837. Dub. Hosp. Gaz. 1857.

[2] Dr. Sutton, Med.-Chir. Trans. xlvii.

[3] Dr. Andrew Clark, Trans. Clin. Soc. i. p. 174.   [4] Epist. section 23, xviii. section 30.

[5] "Inventum novum ex percussione thoracis humani ut signo abstrusos interni pectoris
morbos detegendi," 1761. Trans. by Sir J. Forbes, 1824. He describes this state as
having the consistence of cartilage. Scirrhus was the term universally applied by older
writers to pulmonary indurations, however originating, as by De la Böe, Sylvius, and
Bonetus. (See Waldenburg, "Die Tuberculose," pp. 30, 31, 42.) Avenbrugger does not seem
to have described tubercles, though they were recognised before his time. Avenbrugger's
commentator (Corvisart) has left almost as complete a description of the symptoms as
any subsequently furnished.

[6] Phthisie Pulmonaire, p. 209 et seq. The first two cases are typical illustrations of
this state, and were evidently, from Bayle's description, associated with dilatation of the
bronchi. Bayle considered melanosis to result from a diathetic disease (loc. cit. 84).

[7] Forbes' Trans. 2d Ed. 1827, p. 112. Laennec's description of melanosis of the lung,
under which title he also included melanotic tumours, contains one case of chronic black
induration, associated with tubercle (Ib. p. 390).

[8] The cases included in this analysis will be enumerated in the Appendix at the end of

Pneumonia," " Cirrhosis," " Interstitial Pneumonia," or " Induration of the Lung," which are all that I can find in modern medical literature capable of throwing any light on the general bearings of this question. Many of these are more or less imperfect in regard to history or to pathological details, so that the facts thus gained are only of comparative value. As far, however, as they are available, I shall give the results in a numerical form.

Sex and Age.—Of these cases twenty-two were males and sixteen were females. In one case the sex is not mentioned. The *ages* at which death took place in thirty-eight cases are given in the subjoined table ; but the smallness of the numbers involved and the uncertain duration of the pulmonary affection in many cases, greatly diminishes the value of these results. They show, however, that the disease materially shortens life, since nearly two-thirds of the patients died before attaining the age of forty. (*See* PROGNOSIS.)

AGES AT DEATH.

1 to 10	10 to 20	20 to 30	30 to 40	40 to 50	50 to 60	60 to 70	70 to 80
1	5	10	6	4	9	1	2

The great difficulty in the recognition of the true pathenogenesis of pulmonary indurations arises from the occasional impossibility of determining the origin of masses of cicatricial tissue in the lung, when all signs of the affection in which it originated have passed away. A cicatrix is not a disease, but represents the cure of a past disease, and it is only by a knowledge of the diseases which commonly produce such changes in this organ and of their attendant circumstances, that we can form any conclusion as to the probabilities respecting the antecedent conditions in which it may have originated.

this article. Both in the Appendix and in reference to special points, I have marked such cases by * ; cases not so included I have marked in my references by ‡. I have not included thirty-four cases tabulated by Dr. Sutton as instances of " fibroid degeneration of the lungs " (Med.-Chir. Trans. xlvii.), nor thirty-five cases of bronchial dilatation described by Biermer, many of which presented similar alterations (Zur Theorie und Anatomie der Bronchien-Erweiterung, Virch. Arch. xix.). Both these and Dr. Sutton's cases will be alluded to separately. In addition to these I have only been able to find fifty further cases where any allusion is made to this affection. Many of these are wanting in necessary details of history, or in descriptions of the other lung, or of other organs. Some which relate to cases of recovery, or which illustrate special points, will be again alluded to. I have not, however, included cases *described* as tubercular, but only such published as cases of " cirrhosis," " induration of lung," " interstitial pneumonia," or " chronic pneumonia," and Dr. A. Clark's published case of " fibroid phthisis." I have thought it best to retain in this category some of the cases which appear to me to have been tubercular in their nature, although not described as such, in order to express more clearly the fallacies inherent to this branch of the subject. It must, however, be noticed as remarkable from these numbers how very rare this affection is when uncomplicated with tubercle ; and even some of the cases included in this analysis appear to have had a tubercular origin, or to have been thus complicated. Chomel based his description of the disease on eight cases, including two of his own, which were all that were accessible to him.

There can however, I think, be little doubt that in the majority of cases of induration of the lung found *post mortem*, whether occurring in isolated patches or extending over very considerable areas, the cause lies in the presence of tubercle and of tubercular pneumonia—using these terms in their wider sense to include all forms of granulation ordinarily described as tubercular, and also most of the cheesy changes found in the lungs.[1] This condition has been long recognised, and the fibrous or indurating termination of tubercular processes has been fully described in most works on Pathological Anatomy.[2] So commonly is tubercle found as a complication of this state, that out of four cases quoted by Steffen[3] as examples of "Interstitial Pneumonia," three are most probably tubercular, and the fourth is not free from a similar suspicion. Out of the thirty-four cases of "Fibroid Degeneration" given by Dr. Sutton (loc. cit.), I should regard fifteen at least as presenting similar evidences of indurating tuberculosis, and eleven more as probably having been produced by the same condition, inasmuch as they presented this state as a double affection of both apices associated with cavities,[4] making a total of twenty-six. If indeed the indurated grey granulations, whether occurring singly or in masses, are as Andral thought, the result of Chronic Pneumonia,[5] this hypothesis vastly extends the range of this affection; but this theory of their inflammatory origin is, I believe, just as applicable to the nature of tubercle in general as it is to this special form in which it is sometimes found *post mortem*.

Even out of the thirty-nine cases which I have analysed, I regard

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[1] The cheesy concretions formed by inspissation of puriform matter in the bronchi are, in my experience, much less frequent than is sometimes supposed.

[2] See especially Rokitansky's work. Sir D. Corrigan speaks of "cirrhosis" representing a species of cure for tubercle. See also Cruveilhier, "Tubercles de Cicatrisation" (Anat. Path. liv. xxx. pl. iii. p. 6). He also gives a case where the whole of one lung was indurated by chronic tuberculosis (Anat. Path. Gén. iv. 631). In some cases, however, a microscopic examination will reveal, in cases of fibroid induration, evidences of tubercular growth which are undiscoverable by the naked eye. I have recently observed this in a case which clinically, as well as in the post-mortem appearances, presented a most typical apparent example of "cirrhosis," in the retraction and induration of nearly the whole of one lung with only a small nodule of induration in the other.

[3] Klinik der Kinderkrankheiten.‡

[4] It is undoubtedly true that bronchial dilatations may lead to secondary ulcerations in indurated tissues, but the proportional number of these when independent of tubercle is strangely small when compared with those given by Dr. Sutton. See especially Biermer's paper on "Bronchial Dilatation." Out of thirty-five cases, only twelve were associated with ulcerations of the bronchial mucous membrane, and of these seven were tubercular; while of the five remaining, two were examples of gangrene, and another was a case of abscess of the lung communicating with the bronchi. Barth also considered ulcerations of the bronchi as being very rare, having only met with three instances out of sixty-two cases. The possibility which may be argued that cavities may arise from obstructions of the bronchi, only rests upon what must, when actually tested by observation, be regarded as an exceedingly small number of cases.

[5] It is under this title that Dr. Sutton describes most of these granulations, and he attributes the same opinion to Dr. Addison. Dr. Sutton has, however, carefully distinguished these cases, and has thereby avoided the confusion which might otherwise be caused in pathological descriptions when such a reservation is not adopted. It must be remembered that Andral regarded cheesy matter as the type of tubercle, which he believed to result from an inspissated secretion.

eleven to have been thus associated. In four cases tubercles were found in both lungs; in four others, where the whole of one lung was indurated, they were found in the opposite lung, and in three they were found only in the affected side. Of three cases published by Sir D. Corrigan as instances of "cirrhosis," one was regarded by him as coming under this category, inasmuch as there were cavities in the affected side, and tubercular ulceration of the intestines.[1] Dr. Walshe also alludes to the possibility of "cirrhosis" complicating tubercular disease of the lungs.[2]

The question respecting the other pathological relations of this condition is, however, a complex one, and may be conveniently discussed under the following heads:—

(*a*) The evidence in favour of its origin either in Acute Primary or Broncho-Pneumonia.

(*b*) The evidence of its origin in inflammation of the pleura.

(*c*) The evidence of a simple chronic inflammatory action of the interstitial tissue of the lung, not preceded by either of the above-named acute conditions, and therefore akin to cirrhosis of the liver, or to the granular condition of the kidney.

(*d*) The evidence of an idiopathic "fibroid change" in the walls of the alveoli occurring independently of inflammatory action.

(*a*) The possibility of the origin of fibrous induration of the lung from an attack of Acute Primary Pneumonia is conclusively shown by a case of Andral's,[3] where the acute attack had occurred eighteen months previously, and where after death the lung of the affected side was found universally indurated and traversed by dilated bronchi, in the walls of which a gangrenous action was taking place. The opposite lung was in a condition of recent hepatization; the other viscera were healthy. In addition to this instance, five other cases among those analysed present a similar history, making a total of six, and seven others afford a strong suspicion of a similar origin. Thus, of the only three cases published by Sir D. Corrigan with post-mortem results, one began with an attack of influenza, and in another (the tubercular case before alluded to) the disease appears to have originated with a catarrh, attended with severe pains in the side. Similar evidence is also afforded by three cases reported by Weber[4] of children whom he had himself treated previously for Pneumonia; and he states that he was acquainted with two others still living, who, after attacks of Pneumonia, retained for years the physical signs of induration of the lung, with dilatation of the bronchi; and a similar origin is shown in cases reported by Ziemssen,[5] Reinhardt,[6] Dr. Addison,[7] and Biermer.[8]

[1] Dubl. Hosp. Gaz. 1857.*   [2] Dis. of Lungs, p. 407.
[3] Clin. Méd. iii. obs. 64, p. 474.*   [4] Path. Anat. der Neugeb. ii. 58.‡
[5] Pleuritis und Pneumonie in Kindesalter, p 257.*   [6] Ann. der Charité, i.‡
[7] Collected writings, p. 45.‡   The second of Dr. Addison's cases.
[8] See cases i.‡ and xviii.‡

The conditions of Catarrhal or of the Secondary Broncho-pneumonias, which are more liable than the acute disease to lapse into a chronic state, appear, however, to be more favourable for the production of this change, and it is not improbable that some cases of induration of the lung with dilated bronchi may owe their origin to this form of the disease. Bronchial dilatation is a common event in the Broncho-pneumonia of children, and this condition may persist in cases where the pulmonary consolidation, instead of resolving, passes into a condition of induration. This is shown conclusively by a very instructive case by Bartels,[1] and by two others reported by Dr. Bennett.[2] Another is afforded by Dr. Addison,[3] where the induration of the lung, associated with dilated bronchi, commenced with hooping-cough. Two others with less details are given by Steiner and Neuretter[4] as secondary to bronchitis, and Barth's[5] fourth case is probably an example of the same kind. If we consider the course of acute bronchitis in children, and recollect how constantly dilatation of the bronchi occurs in this condition, both in the idiopathic form of the disease and also in the course of measles and hooping-cough, it can only be a subject of surprise that permanent lesions of this nature are not more commonly met with as the results of these diseases. It has been already stated that the Pneumonia which attends them has a more prolonged course and undergoes a more protracted resolution than is observed in the typical forms of the primary disease ; and it is probably owing in no small degree to the higher reparative powers of childhood that such indurations do not more commonly occur as the sequelæ of these affections. Two cases by Legendre[6] might indeed give rise to the question whether collapse of the lung, together with bronchial dilatation, may not subsequently lead to induration of the pulmonary tissue independently of pneumonic changes, particularly when we recall the statement of Rokitansky,[7] that fibro-nuclear growth in the alveolar walls tends to occur in cases of collapse of long standing.

The mere existence of bronchial dilatation, however acquired, appears to afford a predisposition to pneumonic changes, and to thickening

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[1] Virch. Arch. xxi. p. 144. ‡ This case, where Pneumonia of the apex succeeded to measles, showed in the course of nine months some improvement in the physical signs, but persistent dulness remained at the apex, with signs of dilated bronchi.

[2] Rep. City of London Hosp. for Dis. of Chest.‡ I have not been able to gain access to the originals of these cases. They are quoted at length in the "Journal für Kinderkrank." 1858, p. 305. In both, persistent signs of consolidation of the lung succeeded to measles, and in one case lasted nearly four years.

[3] Loc. cit. p. 44.‡ The first of Dr. Addison's three cases of induration of the lung.

[4] Padiätrische Mittheilungen, Prager Vierteljahresch. 1864, lxxxii. p. 22.‡

[5] Loc. cit. p. 501.*

[6] Rech. Mal. de l'Enfance, 223-283.‡ It appears, however, most probable from Legendre's descriptions, that these changes had been the result of a partially diffused Broncho-pneumonia. He applies to them the term "carnisation," which, from its undefined meaning, has been a frequent source of confusion. Two cases of a very similar nature are cited in Legendre and Bailly's original papers on "Collapse" (loc. cit.). It is possible that they are identical with these.

[7] Path. Anat. 1861, iii. 50.

around the bronchi, which may well explain a large proportion of the instances where these conditions co-exist, and when no definite history of their joint origin in a single attack of an acute affection can be obtained. The progress of interstitial thickening does not, however, appear to affect in this manner large tracts of lung when uncomplicated by other changes, though in some instances it extends inwards, through the interlobular septa from the pleura.[1] There can be no question that bronchiectasis and induration of the pulmonary tissue may reciprocally act as cause and effect to one another, and also that the process leading to induration may simultaneously give rise to dilatation of the bronchi. This explanation, however, fails to explain instances of chronic bronchitis when the clinical evidence of induration of the lung would show that this change is of more recent origin than the cough and expectoration, which in some cases date from an earlier period; and for these I think that the theory of a pneumonia secondary in point of time to the bronchial dilatation affords the best elucidation. The frequency with which such secondary pneumonias occur is variously estimated. Biermer's cases show that they were found in twelve out of fifty-four cases; Rapp (quoted by Biermer) found them in twenty-one out of twenty-four cases; and Barth in twelve out of forty cases.

The pneumonia attending bronchial dilatations is also commonly of the disseminated catarrhal type. It tends especially to occur around the dilatations, when it is frequently set up by the irritation arising from the retained and decomposing products of secretion, or by the direct extension of ulceration or inflammatory action through the bronchial wall. Such forms of Pneumonia are very liable to pass into gangrene, but where this is not the case, the persistence of their cause tends to diminish the possibility of a speedy resolution, and to produce fibrous thickening. Of this tendency several recorded cases afford very good illustrations, which may be regarded as almost conclusive of the nature of this process.[2] Pneumonia having this origin is insidious in its invasion, and does not produce the marked symptoms ordinarily presented by the acute form; and this probably explains some of the reported cases where the commencement of the induration cannot be referred to any single acute attack.

It will readily be understood that when this process has once been established, and when Pneumonia ending in induration has attacked a lung the subject of bronchiectasis, it tends to recur and to repeat itself in other parts of the same organ. The dilated bronchi surrounded by indurated tissue, being a *locus minoris resistentiæ*, are continually liable to become the seat of fresh catarrhal inflammation, from which the process extends to other divisions of the bronchi in the same lung. These in their turn excite disseminated pneumonic changes, which are again prone to the same indurating process. The

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[1] Biermer, loc. cit.

[2] See Case. iv. of Dilatation of Bronchi, by Laennec ‡ (loc. cit. 113); also Biermer's ‡ Obs. i. ii. xiv. xviii. xxi. xxiii. xxiv. xxix. ; also Dr. Stokes, Dis. of Chest, p. 159.*

disease thus' tends to progress *saltatim* until the greater part of the
lung is invaded. Bronchial dilatation may indeed exist, and appa-
rently long, without giving rise to other changes than those caused by
the compression which is produced by the enlarged tubes encroaching
on the surrounding tissue, but the proportion of cases in which this
state is found to exist alone and without attendant induration is com-
paratively small, amounting to only eleven out of the thirty-five cases
reported by Biermer.

The unilateral character of these pulmonary indurations, which
forms a remarkable feature in the history of the disease, and to which
allusion will again be made, may also be compared with the frequency
with which bronchial dilatations are found limited to one lung.[1] The
frequent coincidence of the two affections is also very remarkable, for
dilatations of the bronchi are stated to have existed in thirty-one out
of the thirty-nine cases of pulmonary induration which I have ana-
lysed;[2] while, conversely, on analysing Biermer's cases I find that
induration was present in twenty-four out of the thirty-five cases
of bronchial dilatation reported by him; and Dr. Grainger Stewart[3]
also regards it as a very common though not a necessary compli-
cation of this condition.

Lastly in this category belongs a very large proportion of those
cases where induration of the lung is found in patients exposed
by their occupation to the inhalation of irritating particles of solid
matter, such as the Sheffield grinders, stonemasons, miners, potters,
and cotton workers. In some cases even of this class I am disposed
to believe that tubercular changes may play some part in the produc-
tion of the indurations discovered; but in others, and as far as is at
present known, no evidence of tubercle has been shown to exist. It
appears most probable that the passage of these particles into the air-
vesicles, and their lodgment in their walls, set up a slow pneumonic
process attended by a fibrous growth in the alveolar walls and septa, by
which the indurations observed are produced. These diseases form a
class which requires a separate consideration, but their relation to the
origin of chronic pulmonary induration is of no small importance in
their features of pathological affinity.[4]

(*b*) Pleurisy again seems to be in some cases the exciting cause of this

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[1] Barth says that out of forty-three cases of bronchiectasis the affection was unilateral
in twenty-seven. Biermer says that it occurs with about equal frequency as a double or
as a one-sided affection (Virchow's Handbuch, v., section i. 245). This is probably in
part explicable from the number of cases in which it originates in pneumonia, pleurisy, or
collapse. See Laennec's first case (loc. cit. p. 110‡), where unilateral bronchiectasis re-
mained as the result of hooping-cough.

[2] In two others there is no sufficient account. In six only is this condition stated to
have been absent.

[3] On Dilatation of the Bronchi, 1867.

[4] For an almost complete series of references to the literature of this subject, see
Zenker, Die Staubinhalations Krankheiten der Lungen, Deutsche Arch. Klin. Med. vol.
ii. Also ib. Seltmann, Anthracosis der Lungen. See also Peacock, Brit. For. Med.-Chir.
Rev. xxv. 1860; Dr. Greenhow, Path. Soc. Trans. xviii. xx.; Dr. Hall, Brit. Med. Journ.
March and April 1857; Calvert Holland, Edinb. Journ., 1843.

condition. On ... and Biermer[1] give ... produced is ... Pneumonia may ... pleuritic affec... but it may all ... the case, there ... pretty clear evi... the subseque... extend from the ... do not think that ... surface of the ... walls of the ... Pneumonia

(c, d) If no... existing ... of cases in ... limited. The ... affections of ... antecedent ... twenty-six ... of the other ... many of these ... tion by the ... of Broncho-... will become ... been considered

Mesura ... Grey Indu... presenting ... acute sta... recently ... some ... some fine ... may, how... section ap... tint, but ... passing into ... a gradu...

...ro not densely massed, ...vinced himself that the ... change, and my own ... the nuclei of the alveolar ...lements. In tubercular ...manner similar to those ...ffering from the ordinary ...the other hand they may ...characteristic of tubercle. ...may be found proceeding

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...ch. 1856, vol. xli.). ...us induration, but his ...though the tissue was

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[2] Can...
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induration in this state depends on a gradual thickening of the walls of the air-vesicles,—a thickening which is commonly found in large tracts of the forms of Pneumonia associated with tubercle, as well as in the simpler forms. I have met with this chronic red induration of the base in one case only, and in this there were also masses of tubercular induration in the apex of the same lung, the other lung being free. The patient was an old woman with syphilitic cicatrices in various parts of the body, and a history of earlier syphilis. She had had hæmoptysis seven years before, and no distinct history could be obtained of the date of the invasion of the Pneumonia, but she was under observation for three and a half months with the physical signs of consolidation of the base. Pericarditis with effusion formed the immediate cause of death.

Fig. 1.

The bronchi were dilated in spots of cicatricial contraction of the apex where indurated tubercles were present, and also in the tract of red induration at the base. This tract (Fig. 1) showed on microscopic examination a dense fibre tissue consisting of a network interlacing in all directions, thickening the walls of the pulmonary alveoli, and spreading in all directions through them (a, a). The contents of the alveoli (b, b) were round nucleated cells mostly resembling the pyoid forms seen in the third stage of Pneumonia, but mingled with occasional epithelial cells, and with granular corpuscles and free fat granules. In places (c, d) the air-vesicles are seen to be almost obliterated by this growth, and in some tracts scarcely any traces of them were discoverable. There was comparatively little nucleated growth discoverable in the walls in this case. The process in this condition appears to be only slowly evolved; the growth and thickening of the fibres is gradual, and a rapid development of nucleated cells is not discoverable. In the earlier stages, however, this is sometimes seen as figured in Figs. 2 and 3.

The fibro-nucleated growth is commonly, as is seen in these figures,

in the form of elongated fusiform cells. They are not densely massed, as in the tubercular growths. Heschl has convinced himself that the nuclei of the capillaries participate in the change, and my own observation would confirm his, inasmuch as all the nuclei of the alveolar wall appear to multiply, and to yield fibrous elements. In tubercular indurations, the process may take place in a manner similar to those above described, and without any growths differing from the ordinary fusiform cells of the fibro-plastic type, or on the other hand they may be associated with a dense growth of nuclei characteristic of tubercle. In some cases, indeed (see Fig. 4), the two may be found proceeding
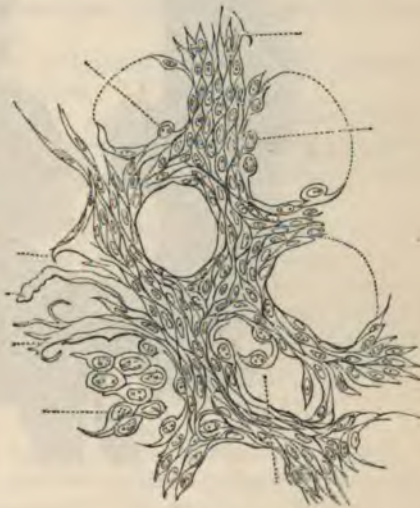
FIG. 2.　　　　　　　　　　　　　FIG. 3.

Fig. 2 is from a case of chronic grey induration associated with tuberculosis, but without tubercle in this part. The contents of the vesicles are an amorphous exudation with few cell-forms.

Fig. 3 is from Heschl (Lungen Induration, Prager Vierteljahresch. 1856, vol. xli.). This is given by Heschl as the mode of growth of the dense fibrous induration, but his case also presented some reddish grey and rusty granulations, though the tissue was indurated to the consistence of fibro-cartilage.

side by side, and occasionally it may even be doubtful what the destination of the nucleated tissue thus originating may be, and whether it shall ultimately form a fibre tissue, or a tubercular mass. The latter, indeed, may finally shrivel by a species of fibrous transformation, or it may be the seat of cheesy transformation or softening leading to the naked-eye appearance of scattered yellow cheesy masses in the midst of indurated tissue. I have only alluded to this mixed growth as an illustration of the frequency with which this combination occurs. With the exception of cases where tubercle is mixed with the indu-
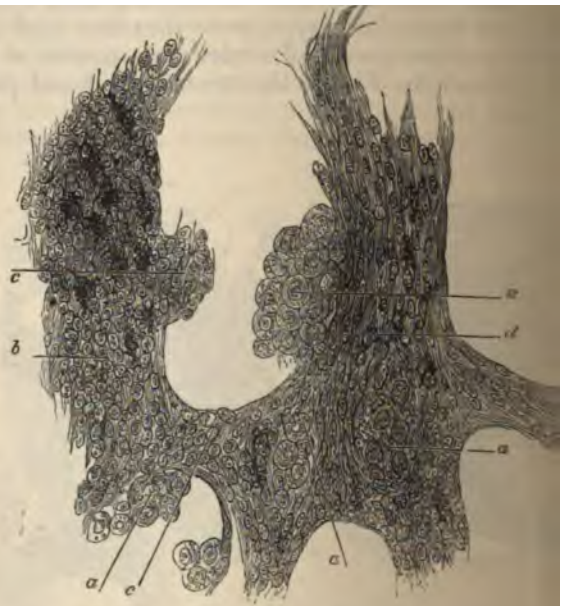
FIG. 4.

Mixed tuberculous and fibro-plastic growth. *a, a, a.* Alveoli fille[d]
epithelial products. *b.* Recent tuberculous growth of round nucle[i]
a fine alveolar network, mingled with masses of pigment. *c.* The sa[me]
the interior of an alveolus. *d.* Fibro-plastic growth of fusiform an[d]
cells. *e.* The same mixed with round nuclei like the tubercular mass.

Other authors have described the induration of the lun[g]
ing on an infiltration of an amorphous substance betwe[en]
stices of the alveoli. This is, I believe, the condition

have failed to show this condition. In
anterior to the use of the microscope, and
mance is found in large tracts of mineral
ening and fusion of large tracts of fibrous
cartilaginous material, closely uniform
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In Chronic Tubercular
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is variously pigmented, and the irregular dissemination o
colouring matter among the white fibrous growth gives it a
grey appearance, which is very characteristic. The alveolar te
the lung is entirely destroyed, though portions may still b
which show traces of pulmonary tissue, and representing earli
of the process. In general, however, the indurated parts, exce
occurring around dilated bronchi, are pretty sharply circum
and the change is usually lobar, or it affects the greater part
or the whole of one lung.

The state of the bronchi in the affected lung is somewhat
In the majority of cases they are dilated, this condition bei
tioned in thirty-one out of thirty-nine cases. In eight o
negative stated. Charcot says that neither in his, nor in Monne
in Hardy and Behier's cases was this dilatation present, but th
to some degree be regarded as exceptional, and Charcot's own ca
to earlier stages of the disease. In some instances, when the
have been found dilated in both lungs, induration has been di
in one only,[1] but usually in such cases the dilatation is gre
the indurated side. In other instances the dilatation has beer
throughout a single lung, a portion of which only has bee
occupied by the indurated tissue; while in the third ar
common form, the dilatation of the bronchi has been limite
indurated part.

The origin of this dilatation of the bronchi has been a su
much discussion. In some it is, as before explained, extrem
bable that it has existed prior to the induration; and in othe
the form of Broncho-pneumonia of childhood, the two may no
quently originate simultaneously. In acute primary Pne
however, dilatation of the bronchi is a rare event, and its ab
probably due to the consolidation of the pulmonary tissue pr
their enlargement within the period at which death usually o
this disease. When the Pneumonia passes into the chron
which is attended with retraction of tissue, various explanatio
been offered of the mechanism of the process.[2] This subject, l
belongs rather to the history of dilatation of the bronchi than
of Chronic Pneumonia. Sir D. Corrigan attributed it to a compe
dilatation of the tubes, in order to fill the space within the
left by the contracting lung. It appears, however, to me to l
probable that the mechanism of this condition is similar to that i
bronchial dilatation takes place under other circumstances, a
it is mainly due to the expiratory force of cough acting on
which in the earlier stages of the disorder are softened, and h:
their elasticity through the inflammatory processes going on i

[1] See Ziemssen's case, before quoted.
[2] Charcot considers that the dilatation of the bronchi in "Cirrhosis" distin:
from Chronic Pneumonia, where he believes it to be absent; but indubitable ev
afforded that it attends induration of the lung secondary to Pneumonia. See
Andral, Weber, and Biermer, before quoted.

It must be remembered that though in the later stages the fibrous tissues formed in this process have a tendency to shrink and contract, they are still deficient in natural elasticity. This defect persists even after they have consolidated into a denser material, and the subsequent contraction would rather have a tendency to diminish the calibre of the bronchi than the reverse. It is less easy to explain the occasional absence of such dilatations, but much would depend on the degree in which the bronchial walls participate in the inflammatory softening, and possibly also on diversities in the rapidity of induration with which we are not yet familiar.[1] In Chronic Tubercular Pneumonia, dilatation of the bronchi is a very common phenomenon, but it is not always easy to decide whether it has been prior or subsequent to the pneumonic changes.

The extent to which this bronchial dilatation may proceed is sometimes very remarkable, and the enlarged tubes may constitute a considerable part of the bulk of the retracted and shrunken lung.[2] The form of the dilatation is not uncommonly globular, and the dilated ends may then form large cavities. It may, however, be simply fusiform. The mucous membrane of the tubes is sometimes smooth; more commonly it is intensely congested, thickened, and villous: in some cases it is ulcerated, but this is rare unless the dilatations are of large size, or except in the presence of tubercle or of sloughing action in the surrounding tissue. Their contents are either the usual muco-purulent secretion, or they may be highly offensive even without the presence of discoverable gangrene in the rest of the lung.

Secondary inflammation in the indurated parts is not uncommon; probably in some instances it extends from the bronchi. It leads to the formation of excavations, and is prone, in some instances, to take on a gangrenous action. Traube, indeed, regards this process as one of the most common causes of gangrene of the lung.[3] In the cases which I have analysed, I find gangrene mentioned twice on the same side as the induration, once on the side opposite.[4] Biermer found gangrene in five out of fifty-four cases, and Barth in three out of forty-three cases of bronchiectasis.[5]

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[1] The same difference exists with respect to the inflammatory softenings of the aorta, which in some cases are the origin of aneurismal dilatations, while in others they indurate without having yielded to the pressure of the blood current.

[2] See a case by Dr. Wilks, Path. Soc. Trans. viii. 39*; also Sir D. Corrigan's cases. This condition is common in the extreme degrees of the affection. The resemblance noticed by Sir D. Corrigan to the bronchi of the tortoise aptly expresses this appearance in many cases.

[3] Deutsche Klinik, 1853-1859. From Prof. Traube's manner of speaking of chronic Pneumonia, he would appear to regard the disease as more common than many other observers do.

[4] Case by Dr. Walshe, Med. Times and Gaz. 1856, i. 156.*

[5] In the thirty-five cases reported by Biermer, gangrene is mentioned in three. In two of these there was induration of the lung. I do not regard bronchiectasis as synonymous with chronic induration, but introduce these numbers for the sake of comparison. Gangrenous Pneumonia may take place in this condition as an acute affection.

[6] In Cruveilhier's Path. Anat. liv. xxxii. is an illustration of this process in a case of chronic tubercular Pneumonia, when a large portion of tissue was separated and lying in

The Pneumonia in other cases, of which four are reported, has led to ulceration of the tissue and the formation of cavities. The total number of cases in which Pneumonia is reported on the same side as the induration is four. In seven others it occurred on the opposite side. In one of these it was gangrenous, and in two others it had led to the formation of abscess.

The pleura is almost invariably thickened, and adhesions to the costal wall are also nearly constant when the disease has made any extensive progress, or has reached the surface. The thickening is sometimes extreme, and occasionally it extends through the interlobular septa into the tissue of the lung.

There is one remarkable feature about this condition to which allusion has been already made, and that is the preponderant number of instances in which one side only has been indurated, amounting to thirty-one out of thirty-nine cases. In five there was Chronic Pneumonia of the opposite side. The whole of the right lung was affected in ten cases, the whole of the left in fourteen, the base alone in eight, and one apex alone in three cases. A double affection of the apex existed in three, but in two of these there is evidence that the affection was tubercular.

Chomel's data are, that out of eight cases, in five the base was affected, in one the whole of one lung, in one the apex, and in one the middle two-thirds of the posterior part of the lung. He states also that in these the bronchi were generally dilated. Durand-Fardel[1] says that in his observations the upper lobe was affected five times, the lower lobe three times, and the middle lobe twice.

The non-affected parts of the lung sometimes present emphysematous changes. This change, usually of the hypertrophous type, is often exceedingly well marked in the sound lung, when one only is extensively affected by retraction and induration.

The bronchial lymphatic glands have sometimes been found to be much enlarged. In other instances they have been simply indurated. When tubercle has existed in the lungs, cheesy spots have in some cases been found in the glands.

PATHOLOGY.—There appears to be but little to add in explanation of cases where the ordinary appearances of pneumonic consolidation in the forms of red, grey, or yellow induration can be traced in direct continuity from a recent but acute attack of primary Pneumonia. Some points, however, require to be noticed with respect to the state of fibrous induration and its relation to other diseases.

a cavity. Cruveilhier does not, however, regard this as a case of gangrene. Dittrich (Lungen-Brand im Folge der Bronchien-Erweiterung) regarded these inflammatory effects as septic, and as arising from retained secretions, and when occurring in the opposite lung, as resulting either from the gravitation of the fluids into the bronchi of the previously sound side, or from constitutional septicæmia. See also Briquet, Mém. sur un Mode de Gangrene du Poumon dependant de la Mortification des Extrémités dilatées des Bronches (Arch. Gén. de Méd. 1841).

[1] Mal. des Vieillards, 601.‡ Durand-Fardel's cases do not all refer to instances of induration.

Addison denied that this state ought to be called a *chronic Pneumonia*, and so far as Pneumonia is a process this criticism is probably correct as applied to the final condition of complete induration, for, as I have before stated, a cicatrised tissue can hardly be termed an inflammatory disease. The question is, however, a different one when we consider the process by which such indurations are produced; and I believe that the evidence which I have analysed will suffice to show that they are very frequently the result of a pneumonia which has passed into a chronic stage.

It remains to be asked whether these indurations result from a process which, as Sir D. Corrigan supposed, has any analogy to cirrhosis of the liver, and from such a condition I believe that sufficient points of difference may be found, to cause serious hesitation in placing the two diseases in the same nosological category.

In the first place, there is this marked diversity between these indurations of the lung and cirrhosis of the liver, that in the lung the fibrous induration of the walls of the pulmonary alveoli is almost invariably, if not constantly, associated with the accumulation of the products of inflammation in the interior of the air-sacs. In a very large number of cases this is demonstrably the result of acute inflammation, and in many more it proceeds, though in a more chronic form, as an accompaniment of the inflammatory process, attendant on the presence of tubercle, or determined by the tubercular diathesis. Further, the change in the liver takes place in a great measure through an increase of the fibrous tissue between the acini; while in the lung, though some thickening is found in the interlobular septa, the most important pathological alterations are those which occur in the walls of the pulmonary alveoli, which certainly have not yet been shown to be the anatomical analogues of the interstitial tissue of a glandular organ, but rather to correspond to the walls of the terminal extremities of the ducts of a gland. Or, to state the difference more briefly, in cirrhosis of the liver the change is external to the lobules and perilobular, while in induration of the lung the fibrous thickening is intralobular. In the liver it is still a question whether the condition known as cirrhosis can be called an inflammation, but in it, at least, there is scarcely any evidence that the cells of the acini of this gland have undergone any changes analogous to those seen in the interior of the pulmonary alveoli.

The granular contracted condition of the kidney, which may be regarded as the most marked analogue of the cirrhotic liver, offers in another respect a striking contrast to these indurations of the lung. In the kidney—a double organ—the affection is almost invariably bilateral, and it is a very rare event to find a single kidney alone affected.[1] In the lung, the double affection is the exception and generally explicable by a tubercular origin, and the single affection is the almost invariable rule when tubercle is not present.

[1] Curiously, a unilateral affection of one kidney has been noticed by Dr. Hilton Fagge, in a case of induration of the lung (Path. Soc. Trans. xx.*) The kidney was partially atrophied in its cortical substance. A calculus, however, existed in one of the calyces.

On these grounds, therefore, I am strongly disposed to
in the vast majority of cases, these thickenings originate
walls as a primary affection, but rather to believe th
almost constant sequence of an alveolar Pneumonia wl
into the chronic stage.

That thickenings of the interlobular septa may a
inwards into the lung as a consequence of chronic ple
doubted fact; but more proof is at present required th
been afforded, that these can implicate the walls of
alveoli to such an extent as to produce a general in
lung with obliteration of the air-vesicles, independen
added pneumonic process, or of the co-existence of tul
only acquainted with two recorded cases which would
out such an opinion.   One is in a note of a post-morter
reported by Dr. Sutton, where it is stated that "sectic
showed that they were uniformly invaded by a tough fil
had destroyed the natural structure and rendered them
and very hard.   There were no circumscribed masses o
is sometimes seen, but the pulmonary texture appearec
parts; thus the natural aspect was lost, being striate
with fibrous filaments."[1]   Parts of the lung were emph
other organs were healthy.   The other case is reported
and Sutton,[2] where one lung only was affected.   Islets
monary tissue appeared among the indurated portions, a
could be seen around the bronchi.

It would require, however, a larger body of proof t
cases appear to me to afford, in order to establish the
independent pulmonary disease, whose essential chara
the thickening of the alveolar wall, as a *primary* affe
independently of inflammatory processes or of tubercul
changes, and it is necessary that this proof should be fu
before such a class can be admitted into our nosolog
I must confess that, though during many years I have
tention to this subject, I have never seen any patholog
supporting such a view, and nearly all the cases of pul
tion which have fallen under my own observation have
with previous Chronic Pneumonia associated with t
tubercles.

instance of "fibroid substitution," it appears to me most important that the inflammatory conditions of its origin should be borne in mind.[1]

The associated pathology of chronic induration of the lung presents some features of interest. The heart is very commonly displaced when retraction of the lung is considerable. It also tends to hypertrophy, but not constantly, as I only find this condition described in eight cases. In four the heart is described as having been healthy. In thirteen its state is not mentioned. In two cases there was contraction of the mitral orifice, and in one tricuspid regurgitation, attended by a characteristic murmur.[2] In one it is described as fatty. In some cases thrombi were found in the pulmonary artery, which under these circumstances has been contracted[3] : Dr. Walshe, however, found it dilated. It may be a subject for further inquiry whether the coagulation of the blood in the branches of this vessel may not in some cases be a cause of protracted resolution of acute Pneumonia, or even of the secondary changes which have now been described. The fact that their mere obstruction may, as shown by Virchow,[4] give rise to inflammatory changes in the pulmonary parenchyma, which are usually persistent, would at least be an argument in favour of this hypothesis. Thickenings have been found in the coats of the pulmonary artery, both by Dr. Schmidt and by Dr. A. Clark. The liver is reported as healthy in eleven cases ; granular and cirrhotic in six ; enlarged and congested in two ; fatty in one ; in nineteen cases there is no mention of its condition. The kidneys were healthy in eight cases ; granular in twelve ; congested in one ; their state is not mentioned in eighteen. The spleen is not mentioned with sufficient frequency to make any analysis useful.

The intestines are commonly reported as healthy : tubercle existed

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[1] The term "Fibroid Phthisis," proposed by my friend Dr. A. Clark, has been very largely debated of late. If it is used to include all diseases tending to produce induration of the lung, it must necessarily comprehend many and widely different pathological processes which conduce to the same result. It is undoubtedly true that the symptoms of "phthisis" may arise from some non-tubercular diseases, and so far the exclusive limitation of the word to tubercular affections may be in a certain sense illogical ; but as in the lung, at least, these form an enormous proportion of the whole, we shall have no option but to retain the term in the present sense, or to fall back upon the heterogeneous classification of Sauvages and Morton ; and the former plan appears likely to be productive of the least amount of confusion in our nomenclature. It is important, doubtless, to recognise the origin of the induration of the lung, and to distinguish the purely pneumonic forms and those which are the result of bronchial dilatation or pleuritic thickenings, from those complicated by tubercle. In the same manner, while recognising the "phthisical" tendency of ulcerative Pneumonia, or of some cases of chronic bronchitis, it would appear more desirable to classify these diseases in their pathological relations rather than in their occasional clinical aspects.

[2] Drs. Barlow and Sutton's case, before quoted.*

[3] Dr. Dickinson, Path. Soc. Trans. xvi.‡ Schmidt, Zwei Fälle von Chronischen Pneumonie. Schmidt's Jahresb. 1866.*

[4] Gesammelte Abhandlungen, 368. One case of Virchow's (loc. cit. p. 274), where old thrombi were found in the pulmonary artery, associated with indurated Chronic Pneumonia, would appear to give a further support to this view. Lebert's and Wyss's experiments (Virchow's Archiv, xl.) on the introduction of solid particles into the circulation have shown that this may give rise to thickening around the obstructed branches of the pulmonary artery, and that such thickenings may extend into the tissue of the lung.

in them in some of the tubercular cases : diarrhœa without tubercle is reported in a few others. Chronic catarrh and congestion of the stomach are reported in a few cases; but the data of a large proportion are imperfect as regards the condition of the gastro-intestinal canal.

When ulceration or gangrenous action has ensued in the indurated parts, metastatic abscesses may be found in other organs. Three instances of this nature are reported where the brain was affected,[1] and another where abscesses of the same kind were found in the liver, spleen, and kidneys.[2]

It does not appear to me, on looking at the general results of this analysis, that the state of the other viscera affords any special ground for the assumption of a " fibroid diathesis" which has been recently maintained to exist as a primary cause of the pulmonary induration. The alterations of the liver and kidneys do not appear to be more common in chronic pulmonary induration than they are in many other chronic diseases, and particularly in those affecting the main conduits of the circulation, whether directly through the heart, or indirectly through the lungs. Both cardiac and pulmonary diseases, which give rise to systemic venous congestion, are liable to cause induration both of the liver and of the kidneys, associated with an increased growth of their interstitial tissue ; and these changes appear to me to be equally common in cases of simple chronic bronchitis and of chronic tubercular phthisis, as in the special affection now under consideration.

There are some other conditions which appear most properly to take their place under the category of Chronic Pneumonia, but which are also of rare occurrence. The chief of these are Chronic Ulcerative Pneumonia and Syphilitic Disease of the Lungs.

CHRONIC ULCERATIVE PNEUMONIA.—The recorded cases of this state occurring independently of tubercular disease are comparatively few. Broussais,[3] indeed, speaks of having met with several, but none are recorded by him except a case of ulceration secondary to the lodgment of a bullet in the lung. Dr. Stokes also speaks of being acquainted with cases of chronic pulmonary abscess arising from Pneumonia, and gives one case where cicatrization had ensued.[4] Bayle,[5] under the title of " Phthisie Ulcéreuse," gives three cases of this nature. The first, of about two months' duration, showed one lung only affected with several ulcerated cavities, the contents of which appear to have been gangrenous. In the second, which was of three years' standing, and where a portion of bone entering the larynx was supposed to

[1] Biermer (loc. cit. p. 244); Lanceraux (Gaz. Méd., Par. 1863); Herard and Cornil (loc. cit.). A very similar case is also reported by Virchow (Archiv für Path. Anat. v. 276).
[2] Lanceraux, loc. cit.
[3] Examen, iv. 156, 336; Hist. des Phlegmasies, ii. 6, note. Broussais here says that during a long period he never met with an instance of this disease uncomplicated by tubercles except when caused by a foreign body in the lungs.   [4] Loc. cit. 316.
[5] Phthisie Pulmonaire, obs. 25 and 26. All the other cases reported by Bayle are more or less complicated by tubercles, but in obs. 28 the only evidence of this consisted in laryngeal ulcerations.

be the exciting cause, both lungs were indurated and contained numerous cavities. Bayle says that he has seen several other cases in which, commonly, there was only one ulcerated cavity. The size of these cavities was sometimes very considerable. Two are reported by him where a large cavity existed in one lung without disease of the other. One of these (Obs. 27) appears to have been a case of secondary ulceration, such as I have before described as occurring in a lung which has already undergone fibrous induration, and the same condition is present in some cases reported by other authors, so that it is difficult to come to a conclusion whether the induration or the cavity formed the primary lesion.[1] A case is recorded by Dr. Risdon Bennett,[2] where the history of the symptoms, which dated from an attack of scarlatina eighteen months previously, would appear to support the latter view, since a large cavity existed at the root of one lung surrounded by a grey infiltration.

The twenty-ninth case recorded by Biermer[3] bears a very close analogy with that last quoted, but here the disease in the lung appeared as secondary to "typhus" (typhoid?), and was only of a month's standing. Numerous spots of Broncho-pneumonia passing into abscesses or forming cavities were found in both lungs. Dilatation of the bronchi was also present, and it may be questioned whether this was not of recent origin, since Buhl has shown that this condition tends to occur under identical circumstances, after continued fever associated with acute destructive Broncho-pneumonia.[4]

There are two fallacies to be guarded against in estimating the pathological significance of ulcerative processes in the lungs, which are, (1) their origin in tubercle, and (2) their origin in pyæmic processes.

The latter need only to be mentioned as a frequent cause of pulmonary abscess, the origin of which it may at times be difficult to discover. It is not unimportant also to remember that hæmorrhagic infarcta may be the cause of indurated spots of cicatricial character, which, after long periods, may show but few traces of their origin.

Ulcerations may also take place from nodules of tubercle situated in the midst of grey or gelatinous hepatization, either recent or of a more chronic and indurated type, and the tubercle, having perished by

[1] See a case by Dr. Green (Path. Soc. Trans. xx.*); also the eleventh case by Barth loc. cit.‡).

[2] Path. Soc. Trans. xii.‡ The sudden expectoration of a large amount of puriform matter in this case led to the suspicion during life of the evacuation of a loculated empyema through the lung, but no distinct evidence of this was afforded by the post-mortem examination. The symptoms and the subsequent expectoration would be quite explicable by an abscess communicating with the bronchi. There was some evidence of a tubercular diathesis.   [3] Loc. cit. p. 274.

[4] Virchow's Archiv, xi. 275, "Ueber Acute Lungen Atrophie." The name does not appear well chosen, since the cases alluded to were those of disseminated gangrenous Pneumonia, following typhoid fever and associated with collapse. Buhl considers that such conditions lead to subsequent shrinking and induration of the pulmonary tissue. The distinction which Buhl establishes for this form of Pneumonia appears to be that it is associated with collapse, and that it passes into acute desquamation and fatty degeneration of the epithelium of the air-vesicles; a process which he regards as being allied to acute atrophy of the liver.

softening, may leave only a cavity surrounded by grey infiltration, or by more or less induration.[1]

SYMPTOMS AND PHYSICAL SIGNS.—A considerable variety has been noticed in these, depending on the stage of the inflammatory action, but still more on the co-existence of bronchial dilatation, or of secondary ulceration or gangrene of the pulmonary tissue, and also on the presence or absence of secondary Pneumonia in the opposite lung.

(a) *In the cases where the state of consolidation has been traced in continuous sequence from an attack of acute primary Pneumonia,* the symptoms present have been chiefly those indicating a prolongation of the pyrexial state, together with a persistence of the physical signs of consolidation of the lung.

The fever does not, however, maintain the acuteness or the typical course observed in the primary disease. In some instances it is scarcely apparent, though the patient remains weak and continues to lose flesh.

In other instances, however, it assumes more of the character of hectic, with irregular exacerbations and remissions, and usually a marked febrile movement takes place towards night. Exact thermometric observations on this subject are wanting, owing to the rarity of the disease in this form. I have already described the characters of the pyrexia in the only case of the kind which has come under my own cognizance. Night sweats sometimes, but not constantly, follow the evening exacerbations ; and emaciation may be very rapid.

There is usually dyspnœa, but this is not always present in a subjective form. The rapidity of respiration also remains greater than natural ; but as the pulse is usually accelerated, the degree of perversion of their ratio to one another, witnessed in the acute stage, is not commonly maintained.

Cough may in some cases be slight, in others it is persistent and troublesome, and may cause a return of the pain in the side. The sputa may in some cases retain a rusty tinge—more commonly they are mucoid or puriform, and with the latter character they may sometimes be expectorated in considerable quantities. Hæmoptysis has not been observed at this period of the disease, though it is common when dilatation of the bronchi and ulcerations have occurred.

The physical examination of the chest reveals at this period phenomena differing in little from those observed in the acute stage.

Retraction of the side to any notable degree does not take place until further induration and contraction of the pulmonary tissues have occurred ; but the tendency is shown even at earlier stages by the case already quoted, of recovery after a protracted convalescence.

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[1] A case reported by Charcot as Chronic Ulcerative Pneumonia (loc. cit. Appendix, p. 66) appears to me to be of this character. Tubercle existed in the opposite lung. A case recorded by Louis (Case iii. Phthisis, Syd. Soc. Ed., trans. by Dr. Walshe, p. 19) was considered by him to belong to this class, inasmuch as tubercle was found in a lymphatic gland in the neck. There were, however, no tubercles in other parts of the body.

Respiratory movements are diminished on the affected side.

Percussion gives a toneless want of resonance, which increases in intensity with the progress of the case. Bronchial or tubular breathing, bronchophony or pectoriloquy, with increased vocal fremitus, are the typical phenomena accompanying this state; but in some instances these have been noticed either to be entirely absent [1] or to have only been intermittingly present, alternating at times with an entire absence of breath-sound.[2]

Râles are generally heard during this period. They are commonly subcrepitant, and the fine crepitation of the acute stage does not appear to persist in the chronic form; large bubbling râles are more common, and they may be sufficiently metallic as to simulate the characters of an abscess or an excavation even when none exists. The respiration in the opposite lung is commonly exaggerated.

If the progress of the case is unfavourable, the digestive system suffers, congestion and catarrh of the stomach supervene, and vomiting is occasionally observed. Thirst is a common symptom. Diarrhœa may also be present, without tubercle or ulceration of the intestines. Anasarca and ascites occasionally occur in the later stages,[3] without any appreciable cause, other than that afforded by the disturbed circulation through the lung.

(b) *When the condition has passed into the more advanced stage of induration,* the symptoms present depend, in great measure, on the co-existent conditions. In some cases the cicatricial tissue formed is perfectly quiescent, and life may be long protracted, without much manifest impairment of the general health, and with only a minor degree of dyspnœa on exertion, although the physical signs of pulmonary induration persist. The presence, however, of dilatation of the bronchi, or the existence of ulcerations of these extending into the pulmonary tissue, or the occurrence of secondary Pneumonia, imparts to the disorder a gradually progressive character, which may strongly simulate the features of tubercular phthisis. These correspond closely to the description of the disease furnished by Avenbrugger and Corvisart. Avenbrugger pointed out that want of respiratory movements and of resonance on percussion were the leading physical signs, while the symptoms present chiefly consisted in dyspnœa on exertion and distension of the jugular and external veins; cough being unfrequent, expectoration scanty, and the decubitus of the patient remaining unaffected. Corvisart, in his commentary, adds to these symptoms a progressive emaciation, and also a febrile diathesis, occasional partial perspirations, loss of appetite and

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[1] Requin, quoted by Grisolle, p. 340. Chomel, loc. cit. 277.

[2] Charcot, loc. cit. p. 39. Charcot only mentions the disappearance of the breath-sound, and not of the other phenomena. Neither he nor Requin have described the state of vocal fremitus. Charcot regrets that the relation of these phenomena to the expectoration was not noticed. It may be remembered that the same condition is sometimes observed in the acute stage. It has also been noticed by Bamberger over bronchial dilatations: Oest. Zeitsch. 1859 (Charcot).

[3] Durand-Fardel, loc. cit. 608.

of sleep, paroxysms of dyspnœa, and, in rare instances, œdema, which occasionally is limited to the limbs of the affected side.

When the complication of *bronchiectasis* is absent, the contraction of the indurated pulmonary tissue produces a gradual retraction of the chest wall on the affected side, which is general when the whole of the lung has been affected, or partial in the upper or lower parts of the chest, according to the site of the induration. If the affection is extensive, displacement of the heart occurs either upwards when the consolidation is seated at the apex of the lung, or if the affection be general, or implicates a large part of the base, the heart is drawn towards the affected side. The contraction of the side has been stated by Dr. Stokes to be as great as that following pleurisy, with the same approximation of the ribs, and procidentia of the shoulder. Dr. Walshe, however, denies that this form of retraction is produced by simple " cirrhosis."

The respiration in some cases, when there is no dilatation [1] of the tubes, has been observed to be bronchial; but there are very few, if any, authentic records of the physical signs in this state. Weak or suppressed breathing must be admitted as being *à priori* possible. We have, I believe, no data respecting the condition of the vocal fremitus and resonance in this condition.

(c). *When dilatation of the bronchi co-exists with chronic pulmonary induration,* many variations occur both in the symptoms and in the physical signs. As a whole, as before stated, they closely simulate those of tubercular phthisis; but the progress of the disease is usually slow, and the deterioration of health and strength proceeds rather through a series of exacerbations than by any marked continuously progressive disease. Dyspnœa is an almost constant, though not an absolutely invariable symptom. The decumbency (when mentioned) is commonly on the affected side. The pulse respiration ratio does not appear to be necessarily or notably perverted.[2] Cough is usually persistent, and is liable at times to marked exacerbations. It may be dry, as originally noticed by Avenbrugger and Chomel, but more commonly it is attended by expectoration.

The sputa, when present, are variable in their characters : sometimes they are simply mucoid; more commonly they are puriform. Under the influence of intercurrent Pneumonia they may become at times rusty in tint. When ulceration is proceeding they acquire a brick-dust appearance, and under these circumstances they are often profuse. They often present a dirty greenish grey appearance, which may approach a bottle-green or inky tint,[3] and they are either confluent, or consist of floating masses of irregular outline, marked by

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[1] *E.g.* in Dr. Andrew Clark's case. Respiration was bronchial under the clavicle of the affected side, where there was only a "thickened sub-pleural nodule," and where the lung tissue otherwise appeared healthy. There were, however, cavities in the central parts of the lung.

[2] Dr. Walshe's case.

[3] Traube (Deutsche Klinik, 1859, 477) considers these characters, and particularly the dark specks, as sufficient to distinguish the sputa of Chronic Pneumonia from those of

black specks and spots of the size of a millet or hemp seed, and they may contain a large amount of pigment and fragments of the elastic tissue of the lung.[1] The colour of the sputa described by Traube has also been noticed by other observers. That these characters are common to dilatation of the bronchi is apparent from Barth's description[2] of their appearance under these circumstances. In one of his cases, where there was a "black softening" of the pulmonary tissue, they were of a chocolate tinge.

Fœtidity of the sputa and also of the breath is a very frequent accompaniment of this condition. I find it mentioned in eleven out of the thirty-nine cases which I have analysed. In four of these it accompanied gangrene of the lungs, but in five others there was no evidence of this state. In two cases, however, the data are imperfect. This offensive character of the sputa may indeed co-exist with simple bronchitis or with bronchial dilatation without pulmonary induration; but the latter is the most common condition in which it occurs, particularly when ulceration and the formation of cavities[3] have taken place.

Hæmoptysis is comparatively a common symptom. I find it mentioned in sixteen out of thirty-nine cases; in seventeen the data are imperfect; in six others its absence may be reasonably inferred. In six cases it was the first symptom that attracted attention, though cough had existed in some antecedently; in two, however, of these there is evidence of tuberculosis, and tubercles were also present in three other cases in which it appeared in the course of the disease. In nine others there were either ulcerations or cavities present, and in two only (in both of which it was slight) are neither of the above conditions recorded. In some cases where it appeared early, it is possible that the ulceration of previously dilated bronchi may have been the cause of its appearance, and that the Broncho-pneumonia thus excited may have led to the subsequent induration of the lung, since in most of these, many years intervened between its appearance and the final fatal issue.

The amount of blood expectorated varies considerably. In the majority of cases it has been moderate, but it is occasionally repeated,

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tubercular phthisis, which he says are masses of yellow or whitish colour, float in water and keep their round shape, and do not present these black specks. The sputa of phthisis are, however, more varied in their appearance than those here stated, and masses of pigment are not, I believe, uncommon in them.

[1] Dr. Walshe.   [2] Loc. cit. 524.

[3] See Dr. Laycock on "Fetid Bronchitis," Case iv. p. 27. Fœtidity of the sputa and of the breath, in connexion with chronic pulmonary induration, was recognised as early as by Willis. Dr. Laycock (p. 9) quotes a case by this author, of a dignitary of the Church who was long troubled with this symptom, and in whom some years later the whole of one lung had undergone the change in question. Dr. Laycock distinguishes this odour as fæcal, in contrast to the special odour of pulmonary gangrene. In a chemical investigation undertaken of one case by Dr. Gamgee, the reaction of the sputa was alkaline. I have found the reaction alkaline in several cases of fœtid sputa; in one also, now under my care, presenting the physical signs of the state at present under consideration. Dr. Walshe (loc. cit.) notes that the sputa are particularly fœtid without being gangrenous, though in his case there was gangrene of the opposite lung, but probably of more recent date.

and it may prove the cause of death.[1]　In a case by W. Schmidt,[2] where there was no evidence of tubercle, the patient had had seventy attacks of pulmonary hæmorrhage in the course of twenty-three years.

The physical signs depend on the existence of dilated bronchi and of cavities in an indurated and retracted lung.　The retraction of the side and the displacement of the heart reaches its extreme degree in this condition; and when the right lung is affected, the right ventricle of the heart may be found beyond the nipple [3] on that side. There is an absolute deficiency of expansion, though some elevation movements may persist over the affected lung.　Percussion gives a high-pitched wooden resonance, which may even be amphoric or tubular, when large dilatations occur near the surface, and particularly in the infra-clavicular regions.　The respiration presents in varying degrees the characters of bronchial or blowing.　Bronchophony and pectoriloquy, the latter also occurring in the whispering variety, are most commonly met with over the affected parts.　The vocal fremitus is usually exaggerated, but its absence has been noticed when both bronchial breathing and bronchophony have been present.[4]　Râles of variable size are usually heard over the dilated tubes.　They are commonly large and bubbling, and are not unfrequently metallic or cavernous in character.　When one lung only is implicated, or unless recent secondary Pneumonia has supervened on the opposite side, the unaffected side is generally hyper-resonant, and the increased clearness on percussion may extend across the middle line, so that under the clavicle on the affected side, as was pointed out by Sir H. Marsh,[5] the dulness is greater towards the acromial angle than towards the sternal articulation of the clavicle.　The respiration in the sound side is exaggerated—puerile; râles are only heard here when bronchitis or Pneumonia is present as a complication.　Lividity of the face, amounting to a minor degree of cyanosis, is observed, and distension of the jugular veins appears to be comparatively not unfrequent.　Peculiar white spots on the face have been noticed by Drs. Barlow and Sutton, and by Dr. Andrew Clark.[6]　Gangrene of the extremities has been noticed as a complication in elderly people.[7] Clubbing of the fingers was noticed in a case by Ziemssen.

Pyrexia, though commonly existing only to a slight degree, is more or less present during the progress of these cases.　There are periods when fever is not present, and its occurrence appears to be due either to intercurrent Pneumonia or to ulcerations and inflammatory action in the indurated tissue surrounding the dilated bronchi.　In some cases of long continuance it is noticed as having occurred at variable

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[1] As in Dr. Sutton's second case,‡ and in one by Dr. Foot, Dub. Journ. 1866, xli.* The latter case was probably tubercular, and the former is not free from the same suspicion.
[2] Zwei Fälle von Chronischen Pneumonie; Erlangen, 1863.　Schmidt's Jahrb. 1866, p. 132.*　　　　　　　　　　　　　　[3] Dr. Walshe's case.
[4] As in a case by Dr. Green, Dubl. Quart. Journ. 1846, p. 510.*
[5] Note by Sir D. Corrigan, Dub. Med. Gaz. 1857, p. 284.
[6] Dr. A. Clark considers these to be indications of fibroid degeneration of the skin.
[7] Durand-Fardel, loc. cit. 604.

intervals, alternating with apyrexial periods of considerable duration during the course of many years.[1] A large proportion of the recorded cases show a certain intermitting degree of febrile action, which in some instances has been severe and of a hectic type towards the close of life. In other instances, even under these circumstances the febrile action has been slight or almost imperceptible. Exact thermometric observations appear, however, to be entirely wanting in all the recorded cases of this disease.

Dropsy is a very common symptom. It is mentioned in thirteen out of thirty-nine cases. Its absence is only distinctly recorded or presumably inferrible in eight. It is seldom extensive, and most commonly affects only the lower extremities. It is sometimes associated with albuminuria and casts of tubes, but in other cases the absence of these has been noted.[2] Ascites, as noticed by Durand-Fardel, is extremely rare.[3] Diarrhœa is not uncommon even without ulceration of the intestines. The latter cause, and probably of a tubercular character, existed however in some cases.[4] Vomiting is also occasionally observed. In some cases where it has been severe, albuminuria has been simultaneously present. In other instances it is excited by spasmodic cough, by difficulty of expectoration, or occasionally by the offensive character of the sputa. In a few instances no explanation of its occurrence has been recorded. Emaciation and loss of flesh proceed in some cases to an extreme degree; in others, though cough had lasted long, there does not appear to have been much wasting of the tissues, but there are very few cases which do not present this to some extent.

DIAGNOSIS.—The diagnosis of Chronic Pneumonia rests on the recognition of the consolidation of the lung, with or without the presence of cavities.

As a general rule, the affection being unilateral, we have to deal only with causes which may affect one lung singly; and the diseases with which this state may be confounded are—pleurisy with effusion, or pleurisy with retraction, collapse of the lung, tubercle, and cancer.

The possibility of *Pleuritic Effusion* can only be admitted in the more recent cases when retraction of the side has not already occurred. Under ordinary circumstances in chronic induration, the absence of enlargement of the side, the distinctness of the intercostal spaces, and the presence of bronchial breathing, bronchophony, and increased vocal fremitus, are usually sufficient to exclude the idea of fluid in the pleura. In the rare cases where the respiratory sounds together with the vocal resonance have been inaudible, the mistake has actually occurred,[5] but attention to the state of the intercostal spaces would

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[1] Schmidt's second case, loc. cit.
[2] In Ziemssen's case (loc. cit.) albumen was absent from the urine when anasarca was first noticed, and only appeared later together with casts of tubes. The data respecting the urine are, as a general rule, imperfect in most of the recorded cases.
[3] Loc. cit. 612.
[4] As in one by Sir D. Corrigan, Dubl. Hosp. Gaz. 1857.   [5] Grisolle, p. 342.

probably prevent this fallacy. A further guide in such cases is the position of the heart, particularly when the affection is on the left side. In effusion it is pushed from the dull side. In Chronic Pneumonia without retraction it is not displaced. Depression of the diaphragm would also aid in the recognition of pleurisy. In the majority of cases, however, these difficulties are unlikely to occur.

Pleurisy with retraction of the side may offer greater difficulties, particularly if the indurated lung does not contain dilated bronchi.

In Chronic Pneumonia, however, the retraction is more general than in pleurisy, and is not, according to the most recent evidence, attended with the same degree of twisting of the ribs on their axes, or with the procidentia of the shoulder and tilting outwards of the angle of the scapula, which attends the retraction following pleurisy.[1] Bronchial breathing and bronchophony are common to both affections; and in pleurisy, even signs of excavation may occasionally be simulated by marked pectoriloquy. But such cases, which are the exception in chronic pleurisy, are the rule in pulmonary indurations associated with dilatation of the bronchi; and therefore the existence of signs of cavities, particularly when general, and when associated with large and metallic râles heard over the affected lung, will be strong evidence in favour of the latter condition. The diagnosis will also be aided by the other features of the case. The characters of the expectoration are often peculiar in Chronic Pneumonia. In pleurisy, if bronchitis be present, they are simply bronchitic, and their source may be discovered by the râles in the opposite lung. Hæmoptysis can only take place in *simple* pleurisy under some conditions affecting the opposite lung. If these be undiscoverable, the presumption would be in favour of Chronic Pneumonia. Pyrexia and emaciation are rarely found in simple chronic pleuritic retraction; and diarrhœa is also uncommon except in the later stages of cases complicated with albuminuria. Regarded as a whole, although no single diagnostic sign exists for the discrimination of the two affections, the group of symptoms characterising Chronic Pneumonia are sufficiently distinct to prevent, in the majority of cases, the possibility of this error in diagnosis.

Simple Collapse of the Lung, sufficiently extensive to simulate retraction with induration, can only occur in the adult as a consequence of obstruction of one of the main bronchial tubes. Except from the introduction of a foreign body, this can only occur from external pressure—as from an aneurism or a tumour originating in the bronchial glands—and would then be attended with other pressure symptoms, or by the physical signs indicative of the nature of the disease. Collapse of the lung is further attended with weakened or suppressed respiratory murmurs. Bronchophony, also, is rarely heard over such parts, and would be practically impossible when the collapse originated from obstruction of the bronchi. Signs of dilated bronchi

[1] Dr. Walshe, Med. Times and Gaz. 1856, i. 1858. The author is indebted to Dr. Walshe's masterly analysis for most of the data on the subject of diagnosis.

are also entirely wanting except in the acute form of Broncho-pneumonia. Pyrexia, and the other symptoms enumerated, are also absent in uncomplicated cases of collapse.

Cancer of the Lung produces retraction of the side, and may lead to signs of excavation. In it, however, the displacement of the heart is much less considerable. The dulness commonly extends across the middle line, while in retraction from Pneumonia the sound lung usually encroaches on the affected side. In cancer, also, pressure signs may be present: the excavations are more extensive, and hæmoptysis is commonly more profuse and repeated. The peculiar currant-jelly-like expectoration of cancer has not been met with in any of the recorded cases of pulmonary induration. Pain in the chest is also much more common in cancer than in Chronic Pneumonia; in the latter disease it appears scarcely ever to be a prominent feature. Local limitation to one side, and the absence of secondary cancerous affections, are less certain guides, but they are *pro tanto* in favour of simple induration. The existence of the cancerous cachexia has not been distinct in the cases which I have seen of primary cancer of the lung. Emaciation and pyrexia are common to both classes of disease. The duration of the case, where it has exceeded more than two years, is, according to Dr. Walshe, almost sufficient to exclude cancer; and this, together with the features first enumerated, are adequate for the diagnosis of the two diseases.

The diagnosis from *tubercle* presents considerable difficulties in some aspects of the question. It is, I think, by no means improbable that a tubercular Pneumonia may, under favourable circumstances, produce a local induration of a part or even of the whole of one lung without necessarily entailing a secondary affection of the opposite lung or of other organs. The indurations attending chronic tubercular phthisis are almost, if not absolutely, identical in their nature with those resulting from a non-tubercular inflammation; and the final result in both cases, of cicatricial tissue traversed by dilated bronchi, produces a condition in which the physical signs and symptoms of the two affections are precisely similar. Such cases are, however, exceptional. In tubercle the disease is commonly progressive, and it is only in very rare cases that the opposite lung is not implicated. The difficulty of diagnosis, however, refers rather to affections of the apex than to those of the base of the lung. A double apex affection, attended with the signs of cavities, is immensely in favour of the tubercular origin of the disease. When, as in some instances, the consolidation affects the base of one lung and the apex of the other, this presumption has also the greatest amount of probability in its favour. A unilateral induration of the whole or the greater part of one lung, when the opposite lung is hypertrophous and healthy, affords on the other hand strong evidence against the tuberculous nature of the affection. Neither the presence nor absence of hæmoptysis, of pyrexia, or diarrhœa, afford any material additional

aid, since they may all occur in chronic induration, and may be absent in cases of chronic phthisis, at least during long periods. Fœtidity of the sputa is rare in phthisis, very common in Chronic Pneumonia with dilated tubes; but it may exist in the former and may be absent in the latter. Under all the circumstances to which I have now alluded, the history of the patient may afford some aid. The history of a previous acute attack occurring on the affected side is largely in favour of the diagnosis of simple induration. The existence of an antecedent but long-continued bronchitis, coupled with occasional attacks of pyrexia, and gradually increasing dyspnœa, would lead to the same conclusion; and practically—though some cases have been recorded as errors in diagnosis—the data for the recognition of the disease are usually sufficiently clear to avoid most of the fallacies which may lead to an erroneous conclusion.

PROGNOSIS.—This may be best studied by considering the duration of the recorded cases. Of these the duration in eight was unknown. In four, death appears to have taken place within twelve months from the first serious symptoms, though whether this represents the whole duration of the disease may be considered as doubtful. The shortest recorded period is three months, mentioned in two of Charcot's and in one of Sir D. Corrigan's cases, and both the former appear to have been simply cases of the acute disease running a protracted and fatal course and ending in ulcerative excavation of the lung. In the case before quoted from Andral, where also the pulmonary induration succeeded to an acute attack of Pneumonia, death took place within eighteen months; but in a similar case recorded by Ziemssen in a child, nine years of progressive illness elapsed between the first attack and the fatal termination. In the remainder analysed, the disease was of unknown duration in six cases. Dating from the first symptoms, death took place in four cases within 2 years, in two within 3 years, in four within 4 years, in four within 5 years, in two within 6 years, in two within 8 years, in three within 11 years, and in six cases life was protracted to 14, 20, 23, 27, 34, and 44 years. Excluding the cases of unknown duration — in many of which, however, pulmonary symptoms had existed during some years—and excluding also those where death took place in less than 12 months, we find that fourteen died within 5 years, and twelve, or nearly an equal number, lived for variable, and sometimes for considerable periods beyond this date.

The general conclusion which may safely be drawn from these figures is, therefore, that under favourable circumstances life may be considerably protracted after distinct signs of consolidation have become apparent, and even when there has been an occasional recurrence of threatening symptoms.[1] The possibility, indeed, of a

[1] This is particularly seen in two cases recorded by Schmidt before quoted, where the patients with signs of pulmonary consolidation with cavities were under observation during periods of fifteen and twenty-three years, and where it is stated that the second of these outlived three physicians who attended him. A boy under the care of Dr. Mayne, whose

nearly perfect restoration to health, though a considerable extent, sometimes amounting to nearly the whole of one lung, is rendered impervious to air, may be seen in some of the cases before quoted, especially in children, where the affection has succeeded to an acute attack of Pneumonia or of Broncho-pneumonia. In none of these cases, however, is there any evidence that the affected part ever regained its respiratory function; and it may be regarded as more than doubtful whether organized cicatricial tissue of the lung is ever removed by any process, so as to restore the natural condition. In some cases, where only a part of the lung has been thus indurated, its gradual contraction is compensated for by an hypertrophous emphysema of the remainder; and thus the area over which the physical signs of induration exist may gradually diminish, and may become replaced by those of healthy pulmonary tissue.[1] If the whole of one lung has been indurated, the process of respiration is necessarily confined to the other, and this increases the danger arising from any subsequent disease, by which the sound organ may be incapacitated from performing its functions.[2]

The other elements in the prognosis depend on the progress of the disease in the affected parts, and on the general constitutional state of the patient.

Evidences of chronic inflammatory action in the bronchi, as shown by catarrh, with pyrexia and profuse expectoration, have, in addition to the exhausting effects which these directly produce, the further unfavourable significance, that they threaten an extension of the inflammatory action to the surrounding pulmonary tissue, and are thus a source of danger from ulceration and the formation of cavities. The more quiescent these symptoms appear, the more favourable therefore is the ultimate prognosis.

The existence of ulceration as shown by hæmoptysis, or by the expectoration of grumous fragments of lung tissue, are unfavourable signs;[3] and though hæmoptysis may, as before stated, be in some cases frequently repeated during many years, yet, in the majority of instances, cases presenting this symptom to a marked degree have a more unfavourable course than those in which it is slighter in amount or altogether absent; and it must further be remembered, that such hæmoptysis may occasionally prove immediately fatal. Cases commencing with hæmoptysis are always open to the suspicion of being of tubercular origin, but the very existence of an indurating tendency

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ase is not free from the suspicion of tubercle, lived six years of a laborious life involving great exposure, after distinct disease had been observed in one lung, and died only of acute bronchitis affecting the other. (Dubl. Hosp. Gaz. 1860, vii.*)

[1] As in Bartel's case, before quoted.

[2] As in Dr. Mayne's case (Dubl. Hosp. Gaz. 1860, vol. vii.), where death was caused by acute bronchitis affecting the previously sound lung.

[3] The discovery of elastic lung fibres in these fragments is the only positive evidence of pulmonary destruction; but when ulceration affects an old induration, the elastic fibres may be undiscoverable.

in tuberculous disease imparts to such cases a comparatively favourable prognosis; and some of these, as recorded, appear to have quite as favourable a chance of prolongation of life as those cases where the primary induration appeared to be of simple inflammatory origin.

An offensive character of the sputa almost invariably gives a more serious character to the case; but life may in some instances be long protracted even after this has appeared. Fœtidity of the expectoration may co-exist with simple dilatation of the bronchi, but the character of the secretions in such cases imparts to them a septic tendency, and increases the liability to septic or gangrenous Pneumonia, either in the affected or in the opposite lung. In many cases, indeed, such fœtidity is immediately associated with ulcerative processes, and the recognition of these may probably be aided in great measure by the co-existence of pyrexia with this state.

The presence of fever under such circumstances usually depends on the existence of secondary Pneumonia; and when it is undiscoverable on the sound side, it is a matter of great probability that it is extending in that already affected. Pyrexia, when of long continuance, has been indeed one of the final phenomena in nearly all the recorded cases, and its existence is always to be regarded with the gravest suspicion, both in its diagnostic significance and also through its exhausting effects on the nutrition and strength of the patient.

Emaciation is naturally an unfavourable sign, but in some cases death may ensue without any extreme degree of marasmus being attained.

Dropsy is always an unfavourable symptom. Very few cases attained to any considerable prolongation of life after it had appeared. Even when albuminuria is absent, it is indicative both of serious disturbance of the circulation and probably also of an hydræmic condition of the blood; and the co-existence of albuminuria and the presence of casts of tubes in the urine adds an additional gravity to the prognosis, by their significance as expressions of a general impairment of nutrition.

Diarrhœa and vomiting are also signs of the gravest import. They rapidly exhaust the patient, and in some cases lead directly to the fatal issue. Finally, when ulcerative action is proceeding, the possibility of secondary metastatic affections must also be recollected. In one case, by Andral, sudden death is reported to have occurred without any adequate explanation being afforded of the cause of such a termination.[1]

TREATMENT.—The treatment of Chronic Pneumonia may be considered under two categories. The first includes the cases where the disease has *recently* lapsed from the acute stage. The second com-

[1] The suspicion here may arise of thrombosis of the pulmonary artery.

prehends those where thickening and fibrous growth have taken place in the walls of the air-vesicles.

(*a*) Cases of the first type are often serious in their character, and the continuous pyrexia and progressive emaciation tend to a fatal issue.

The great principle to be followed in such cases is steadily to maintain the strength of the patient, and to meet individual symptoms as they arise.

Sufficient evidence exists to show that under these conditions progressive improvement may be observed, and that the lung may be restored to its healthy state. I have the strongest doubts whether medicinal agents have any direct effect in accelerating the absorption of the exudation matter from the interior of the air-cells of the lung, and I believe that the administration of mercurials, or even of the preparations of iodine with this purpose, is likely to defeat the main object which should be pursued of maintaining and improving nutrition.

As long as pyrexia persists the patient should be kept in bed, in order to economize, as far as possible, the expenditure of strength and the waste of tissue. The diet should be liberal, but proportioned in quantity and quality to the digestive powers; and milk may, when it agrees, be freely taken with advantage. Alcoholic stimulants, in moderation, are required in most cases; the form selected is best determined by their effects on the individual case. It is important to watch their effects on the pyrexia, which should be made the object of careful and repeated thermometric observations. It has appeared to me that they are best given as far as possible during the periods of remission, and that they should be withheld or given in diminished quantities before and during the febrile exacerbations. When night perspirations are present, a moderate dose of stimulant will often have the effect of checking these, and also of obviating some of the exhaustion which is felt on the succeeding morning.

Cough may be allayed by small doses of opiates combined with three or four drops of the vin. ipecacuanhæ, and with from five to ten grains of the muriate of ammonia. Iron, quinine, bark, and the mineral acids may all be employed according to the special indications of anæmia, failure of appetite, sweating, or profuse expectoration. If the latter be present, ipecacuanha should be withheld, and ammonia with infusion of senega or serpentaria may be given. The existence of gastric catarrh often forms an unfavourable complication of these cases. Its presence appears to me to contra-indicate most of the remedies last enumerated, and it is usually aggravated by the whole class of "tonics." If it co-exists with much cough and expectoration, these may be met with opiates and the muriate of ammonia, while the gastric disorder is best treated by bismuth, alkalies, and occasionally by small doses of hydrocyanic acid. Under this management a gradual improvement will often take place, and it has appeared to me that this may be aided by small blisters repeatedly

applied over the affected side—a method which I think better than the use of extensive vesication, which may disturb sleep and exhaust the patient.

(*b*) In the treatment of chronic induration, it is, I believe, best to recognise the fact that fibrous tissue once formed is incapable of being removed by medicinal treatment. The management of such cases is therefore mainly hygienic, and should be directed to maintain the health and to prevent extension of the disease in the affected side or secondary affections of the opposite lung.

The dilatation of the bronchi which commonly attends this condition, renders the patient liable to catarrh; and since it is from repeated attacks of bronchial inflammation that the most dangerous effects of the disease are likely to arise, the avoidance of all causes of this nature is therefore of primary importance. Flannel worn next the skin, avoidance of exposure, and the resources of a climate suited to the individual case, and which may be in part determined by experience and in part by the conditions of irritability or of more or less tendency to bronchial secretion, are among the most important elements to be considered among the prophylactic agencies.

Every fresh catarrh should be at once and promptly met, at least in its acute stage, by confinement to a regulated atmosphere, by counter-irritation to the chest, and by opiates, ipecacuanha, or muriate of ammonia.

The maintenance of the general health and of the nutrition are also of essential importance. A liberal diet, of which milk may form a large share, and a judicious use of alcoholic stimulants whenever pyrexia is absent, are almost always required. Cod-liver oil may also be taken with advantage during long periods when the digestion will tolerate its use. Iron and bark should be given, if the indications for their administration arise.

If any of the severer complications are present, they should be appropriately treated—hæmoptysis by gallic acid or the acetate of lead or ergot; diarrhœa by astringents; vomiting by small doses of opiates or by hydrocyanic acid; and gastric catarrh by bismuth and alkalies.

Profuse expectoration may be met by inhalations of oleum picis, creosote, or carbolic acid, either in the form of vapour or by means of a weak solution in an atomiser; and the muriate of ammonia may sometimes be used with advantage in the same manner. The inhalation of the vapour of the oil of turpentine has been tried in some cases where the sputa have been fœtid, but unfortunately without much success. The vapour of iodine has appeared to me to give more relief in some cases when gangrene has imparted this character to the sputa. Port wine in full doses often has a most beneficial effect in cases characterised by profuse expectoration, and preparations of bark and the mineral acids have also a favourable influence.

Opiates not only check secretion, but diminish the violence of the

cough, and relieve the bronchi from the continual tendency thus excited to produce further dilatation.

The treatment of albuminuria, dropsy, or ulceration of the pulmonary tissue, when this has occurred, is unfortunately almost beyond the reach of remedial agents; and though much may be effected before these complications have occurred, the later stages of this condition can only be met by such indications as may promote the comfort of the patient, rather than with any hopes of restoration.

APPENDIX TO ARTICLE ON CHRONIC PNEUMONIA.

THE rarity of this disease and the difficulty attending some points of its pathology induce me to believe that references to the most important published cases illustrating it may possibly be useful to others. I shall continue to mark the cases which I have tabulated by an asterisk (*).

Bayle (Phthisie Pulmonaire), two cases of "phthisie avec mélanose."* Laennec (Forbes' translation, 2d Edition, p. 112), a case of dilatation of the bronchi with pulmonary induration.* Andral (Clinique Médicale, iii. obs. lxiv.), a case where induration of the lung succeeded an attack of acute pneumonia.* Dr. Stokes (Diseases of Chest, p. 150), a case of chronic pulmonary induration with dilatation of the bronchi.* Jaccoud (Clinique Médicale, p. 82), sclerosis of the lung with dilatation of the bronchi—*tubercular?** Herard and Cornil (Phthisie Pulmonaire, 167), induration of lung with bronchial dilatation.* Ziemssen (Pleuritis und Pneumonie im Kindesalter), a case of nine years' standing in a child, probably resulting from an attack of acute pneumonia; dilatation of bronchi in both lungs; induration of one.* Barth (Rech. sur la Dilatation des Bronches; Mém. Soc. Méd. Obs. 1856), six cases; * *some probably tubercular.* Heschl (Prager Vierteljahresch. 1856, ii.), two cases: only pathological details.* W. Schmidt (Zwei Fälle von chronischen Pneumonie, Diss. Erlangen, 1863).* Charcot (De la Pneumonie chronique), three cases,* two acute. Dr. Green (Path. Soc. Trans., vol. xx.), ulcerative pneumonia.* Dr. Peacock (Edinb. Journ. 1855).* Raimbert (Journ. Méd. et Pharm. de Bruxelles: analysis in Gazette Hebdom. 1856), one case incomplete; * others referred to are described as "carnification." Dr. Andrew Clark, a case of fibroid phthisis* (Trans. Clin. Soc. i.), *probably tubercular.*

The following are described as cases of "cirrhosis:"—

Sir D. Corrigan (Dublin Journal, 1838, and Dublin Hospital Gazette, 1857), three cases with post-mortem results.* Dr. Walshe (Med. Times and Gazette, 1856), the most fully-recorded case extant with commentary.* Dr. Mayne (Dub. Hosp. Gaz., 1857 and 1860), two cases,* the last *doubtfully tubercular.* Dr. Green (Dublin Quarterly Journal, 1846).* Dr. Law (ib. 1848),* *probably tubercular.* Dr. Jennings (ib. 1866).* Dr. Foot (ib. 1866), *doubtfully tubercular.** Dr. Wilks (Path. Soc. Trans., viii.), *probably tubercular, mentions "spots of strumous deposit" in the opposite lung.* Drs. Barlow and Sutton (Path. Soc. Trans. xvi.).* Dr. Fagge (ib. vol. xx.).* Dr. Barlow (Guy's Hosp. Rep. 1847, 2d Ser. v.).*

The following cases not included in the analysis as being either of doubtful nature or imperfect in general details, may also be referred to:—

Dr. Dickinson (Path. Soc. Trans. xiii.). Dr. Risdon Bennett (Path. Soc. Trans., xii.), a case of ulcerative pneumonia of doubtful origin. Biermer (Zur Theorie und Anatomie der Bronchien-Erweiterung, Virch. Arch. xix.), many instances of induration of lung. Macdowell (Dublin Quarterly Journal, 1856), entitled as "Cirrhosis," but lung described as "carnified;" no dilatation of bronchi; thickened pleura and fluid in pleural cavity. Weber

(Path. Anat. der Neugeborenen und Säuglinge, ii.), three cases referred to where induration of lung commenced with acute pneumonia, with post-mortem results, and two more of recovery. Bartels (Virch. Arch. xxi.), a similar case commencing with broncho-pneumonia. Steffen (Klinik der Kinderkrankheiten), four cases of interstitial pneumonia ; three at least *tubercular*. Legendre (Rech. Anat. Path. Maladies de l'Enfance), two cases of induration of lung in children ; secondary to catarrhal pneumonia. Dr. Bennett (Rep. City of London Hosp. for Dis. of Chest), two cases of induration with signs of dilated bronchi in children ; recovery. Traube (Deutsche Klinik, 1859, § iv.), two cases, chronic ulcerative pneumonia ; general remarks. Dr. Addison's Works (Syd. Soc. Ed.), three cases. Steiner and Neuretter, Padiätrische Mittheilungen (Prager Viertel jahresch. 1866, lxxxii.), two cases of induration after broncho-pneumonia. Macquet (Bull. Soc. Anat. xxii.). Gabalda (ib.). Charnal (ib. vol. xxx.), induration of lung in conse-quence of acute pneumonia ; other data imperfect. Barth (ib. vol. xxix.), induration of base with dilated bronchi. Barset (Bull. Soc. Anat. xxx.), microscopic examination by Robin after maceration. Lancereaux, two cases of gangrenous pneumonia, surrounded by induration, and associated with secondary abscesses in other organs (Gaz. Méd. Paris, 1863). Powell (Trans. Clin. Soc. ii.), cases of phthisis with contracted lung ; excellent description of appearances, all of *tubercular nature*. Sutton, fibroid degeneration of lungs (Med.-Chir. Trans. 1865, xlviii.). Bastian, Cirrhosis of Lungs (Path. Soc. Trans. xx.), tabulated series of thirty-four cases of induration. Durand-Fardel (Mal. des Vieillards), various forms of chronic pneumonia. Cotton on a prevailing form of Chronic Pneumonia (Med. Times and Gaz. 1855, i.) ; general description, pathological appearances of two cases. Hardy and Béhier (Path. Interne) ; general description, ref. to four cases. Grisolle (Traité de la Pneumonie), ref. to various cases. Lebert (Physiologie Patho-logique), ref. to pathological appearances in two cases. Chomel (Dict. de Méd. xvii.), general description. Avenbruegger (Inventum Novum, &c., 1761), "Scirrhus of Lung," with commentary by Corvisart (Eng. Trans. by Sir J. Forbes). Sir J. Forbes (Appendix to Trans. Laennec, Ed. 1824), two cases, one probably tubercular, the other doubtful, tissue of lung floated. Broussais (Hist. der Phlegm. vol. ii.), three cases. Hasse (Path. Anat., Syd. Soc. Ed.). Hope (Morbid Anatomy). Rokitansky (Path. Anat.). Cruveilhier (Anat. Path., liv. xxxii.). In addition to these, the following works, to which I have not been able to obtain access, are referred to by Grisolle, Durand-Fardel, and Charcot, but the data of many appear from the statements of these authors to be unreliable or imperfect :—Letenneur (Diss. Pneumonie chronique, Thèses de Paris, 1811). Bazière (Diss. sur l'Emploi du Séton dans la Pneumonie chronique, 1819). Rat, Thèses de Paris, 1845. Raymond, Sur la Pneumonie chronique simple, Diss. 1842. The author is indebted to Dr. Bastian's tables and to M. Charcot's thesis for several references to cases, some of which are included in the foregoing analysis.

(Since this article was printed I have met with two other illustrations : Dr. E. Long Fox (Med. Times and Gaz. 1870), a case of chronic ulcerative pneumonia —thermometric observations; Immermann (Deutsch. Arch. Klin. Med. V. p. 235 et seq.), a case of chronic pulmonary induration (tubercular ?), attended with stenosis of the pulmonary artery.)

SYPHILITIC AFFECTIONS OF THE LUNG.[1]

By Wilson Fox, M.D., F.R.C.P.

The manner in which the tissue of the lung may be affected by the syphilitic poison, although it has been made the subject of much recent research, still requires a more accurate definition than has yet been attained.

The opinion that certain forms of phthisis may arise from changes in the pulmonary tissue, due to the syphilitic poison, is no new one. Morgagni noticed the frequent connection of tubercle with this dyscrasia, and Portal and Morton described a syphilitic phthisis, but failed to show that any special pathological changes were connected with this condition. Dr. Graves and Dr. Stokes[2] have both entertained a similar opinion, based upon the success of the mercurial treatment of bronchitis in patients who had formerly been the subjects of venereal sores. Bayle, Laennec, and Louis failed to find any evidence of a special form of phthisis which could be distinguished as syphilitic, and it is only within recent periods that any changes have been identified in the lungs, which can probably be attributed to this cause.

The difficulty of the inquiry lies in establishing any certain criteria by which such alterations can be distinguished from the changes produced either by simple inflammatory, or by the tubercular processes. Each of these may affect syphilitic patients, and may run a course apparently unmodified either clinically or pathologically by the specific dyscrasia; and looking at the general history of syphilitic affections, it is at least probable that the lungs are less prone to suffer from secondary or tertiary affections of a syphilitic character than the

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[1] I have not met with any indubitable instances of these affections in my pathological studies on the diseases of the lungs, and the information contained under this head has been drawn from the following authors, in addition to those alluded to subsequently:— Virchow, Archiv, xv., and Krankhaften Geschwülste, vol. ii.; historical data and complete references. E. Wagner, Arch. der Heilkunde, 1863, vol. iv. Foerster, Würzb. Med. Zeitsch. 1863, vol. iv.; Berkeley Hill, Syphilis and Local Contagious Disorders, many references. Von Baerensprung, Die Hereditäre Syphilis: many cases; microscopic figures of gummata in the lungs. Lancereaux, Traité Hist. et Pract. de la Syphilis; extensive bibliography, numerous cases. Lebert, Traité d'Anat Path. Pl. xciii., figures of gummata in the lungs. Wilks, Guy's Hosp. Rep., 1863, and Path. Soc. Trans. ix., figures of gummata in the lungs; also A Lecture on Syphilis. Pihan Dufeillay, Des Dégénérescences Syphilitiques des Viscères, Union Méd. 1861, and in Bull Soc. Anat. 1861; comments in a case of Cornil's; numerous references and critical observations.

[2] Graves, Clin. Med. ii. 27. Stokes, Dis. of Chest, 94—432.

mucous membranes of the upper air-passages, or than the skin, the eye, or the bones. What their comparative liability may be in respect to the liver, the spleen, the testicle, or the brain, is a point which must yet be determined by further research. In the lungs of syphilitic patients which I have examined, I have seen no appearances differing from those of ordinary pneumonia, of ordinary tubercle, or of tubercular or cheesy infiltrations; and one marked case of this kind has come under my observation, where there was the most distinct syphilitic ulceration of the larynx, but where the lungs only presented a grey infiltration, together with tubercles and indurations referable to a previous attack affecting the apices, the cure of which I had myself witnessed at an earlier date. Other instances of an analogous kind have come under my observation, where the most careful microscopic examination failed to reveal any peculiarities which I could ascribe to a syphilitic process.

The inquiry into the nature of changes attributable to syphilis is therefore for the present almost a purely pathological one, though the importance of the question in its clinical aspect can scarcely be over-rated. A large amount of the evidence on this subject is derived from premature or stillborn children, the offspring of syphilitic parents; but some cases are recorded where syphilitic gummata have been found in the lungs of adults.

There are two sets of changes in the lungs, regarding the syphilitic nature of which there is a considerable unanimity of opinion. In another large class there is more doubt as to their true connexion with this poison. The former are at least rare, and only isolated instances are recorded by observers having large opportunities for pathological research. The latter class requires a most careful and critical examination before their specific nature can be admitted.

The most authentic changes in the lungs which can be ascribed to syphilis are gummata, or masses of low fibrous growth, evincing a great tendency to necrobiotic changes of the dry cheesy type, and which are very closely analogous to similar masses found in the liver and in other internal organs. They are found in the lungs of adults, and of newly-born syphilitic children. In the former, however, they are so extremely rare, that Lancereaux has only been able to collect ten cases by different authors. They are irregularly distributed through the lungs, having no special seat of predilection, but according to Wagner they are more common in the deeper than in the peripheric parts. They may be single or multiple, and their dimensions may vary from the size of a pea to that of a walnut, or even of a goose's egg.[1] They are generally rounded, rarely irregular in outline, and are sharply defined, but are not always encapsuled.[2] In their earlier stages they are grey or brownish red, completely homogeneous to the

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[1] E. Wagner.

[2] Ib. (loc. cit.). Von Baerensprung describes smaller masses in the lungs of newly-born children as sharply defined by a layer of well-developed fibrous tissue. The nodules in Dr. Wilks's case do not appear to have been thus encapsuled.

naked eye, and are firm and dryish:—later they become of a comparatively uniform yellowish tint, but still maintaining their dry firm character. In some instances, however, they soften and form actual, or, more commonly, potential cavities.[1] On microscopic examination they are found to consist of imperfectly formed fibres, which are often granular and are intermixed with abortive nuclei and a few fibre cells. Both the nuclei and the cells are commonly found in various stages of fatty degeneration. The lung tissue is entirely destroyed by this growth, by which the walls of the alveoli become progressively thickened, until the cavity of the vesicles is obliterated, while the epithelium lining them appears to participate but little in the change. In some cases the bronchi show an infiltration of the submucous cellular tissue with a fibro-nucleated growth, which may form small prominences on the surface. Similar masses are sometimes found in their deeper structures, but these as a general rule are unaltered. In these changes the preponderant and distinctive character consists in the growth of an indurating fibrous tissue, mingled with abortive nuclei, into distinct masses, and presenting a strong tendency to an early necrotic change.

Another form, termed by Wagner the "diffused," is the appearance described by Virchow[2] and Weber[3] as the "white hepatization of the lungs" of newly-born children: it has also been named "Epithelioma of the lungs," by Lorain and Robin;[4] and its syphilitic character has been shown by the last-named authors, who traced a relation between it and syphilitic pemphigus, and also by Hecker,[5] Howitz,[6] and Wagner,[7] and this has also been admitted by Virchow. Lungs in this state are distended so as to completely fill the cavity of the thorax, and to bear the impress of the ribs. The pleura covering them is usually found unaffected. They are white, dense, firm and hard. They occasionally admit of partial insufflation, but this is not constant. Their weight when the affection is general is four or five times greater than natural. Their colour is whitish with a shade of yellow, and it is uniform without any shading. Their section is smooth and opaque. They are resistant in some cases;—in others, as described by Weber, the finger can be pressed into them as into a fatty liver. They are quite exsanguine, and not a trace of blood or of the smaller blood-vessels can be discovered in them. The lobular texture is apparent—the interlobular tissue sometimes presents a slightly reddish tinge. The bronchi contain a tough mucus. The bronchial glands are enlarged, greyish, homogeneous, or in parts presenting a dry cheesy aspect. The extent of

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[1] Dr. Wilks (loc. cit.). Ricord (Clin. Iconograph. Pl. 28) gives a case where numerous softened masses were found in the lungs, but he questions whether they were not the result of pyæmic infection. Depaul, one of the earliest authors who has published authentic observations on this subject (Bull Soc. Anat. 1837 ; Gaz. Méd. 1851 ; Mém. Acad. Imp. de Méd. 1853), has also found the centre of these masses softening into a puriform fluid, and sometimes presenting real abscesses, whose walls were formed by a yellowish grey and indurated tissue.

[2] Archiv, i. 146.   [3] Path. Anat. der Neugeborenen, ii. 47.
[4] Gaz. Méd., Par. 1855.   [5] Verhand. der Berlin. Geburtshülf Gesell. 1854, viii. 130.
[6] Behrend's Syphiologie, 1862, iii. 611.   [7] Loc. cit.

infiltration varies—sometimes the whole of both lungs are affected,[1] sometimes only parts. When the affection is partial, there may be found in addition to the general infiltration isolated spots of the same kind, but resembling more or less the gummata before described, which sometimes merge at their margins into the neighbouring infiltration. There is some discrepancy between the statements of different observers regarding the histological characters of this consolidation. Virchow described the air-vesicles as filled with epithelial cells, and Robin and Lorain make the same statement, and add that this process extends into the ultimate bronchial ramifications—but that at the same time the walls of the alveoli are thickened and rigid. Weber described the contents of the alveoli as cellular; while Wagner, from his recent researches, says that the characteristic by which this change may be distinguished from grey hepatization is, that nothing can be brushed or washed out from the interior of the vesicles, and that the disease essentially consists in a thickening of the alveolar walls, by which the cavity of the vesicles is gradually obliterated, and that in this process the epithelial lining is but little affected. This thickening takes place by the growth of an imperfect and scantily fibrillated tissue mingled with nuclei, and of a few fibre cells which are found in various stages of fatty and molecular disintegration; granular and fatty débris are also found in large proportions throughout the tissue. The interlobular texture is normal or contains a small amount of nuclear and cell growth. The vessels and capillaries are almost completely destroyed in the affected parts.

The submucous tissue of the bronchi is affected in the same manner as has been described as occurring in connexion with the gummata, by a growth of nuclei limited to the superficial structures.

The bronchial glands are enlarged, and show concentric masses of cells bounded by a tough fibre tissue.

It will be observed that in both of these forms of disease the essential characteristic of the change described consists in a thickening of the walls of the air-vesicles by a growth of imperfect fibre tissue mingled with nuclei which tends to pass into an early molecular detritus, and that this change thus produces a structure apparently identical with the syphilitic gummata found in the liver.

Even in this form it would be very difficult to state any precise definition which might absolutely distinguish the process from the similar changes which occur in tubercular growths, and in the thickenings which affect the walls of the air-vesicles in tubercular pneumonia.[2]

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[1] Wagner in six cases found the whole of both lungs affected four times; once the half, and once the sixth part of the lung. Kostlin (Arch. Phys. Heilk. xvii.) met with it in four cases, generally limited to the lower lobe, or in isolated masses, varying in size from a pea to a pigeon's egg. In one child, who lived a fortnight after birth, the signs of the disease in the lungs appeared coincidently with ecthymatous pustules, with a measly rash, and with excoriations of the skin.

[2] Lancereaux (426) says that large granule cells are not found in tubercular growths; but this distinction is not, I believe, to be relied upon. My own observations on tubercular formations have convinced me that such granular cells are by no means uncommon in these.

This difficulty is further increased in relation to some of the other changes which are frequently found in the lungs of syphilitic children, and also in some cases of adults. These, if separately distinguished, may be enumerated as follows:—

(*a*) Foerster has shown that lobular, vesicular, and broncho-pneumonia, either in a disseminated or in a confluent form, is very common in the lungs of children affected with hereditary syphilis and dying shortly after birth. In the majority of cases such pneumonias are identical in character with the ordinary forms of the disease, and consist only of an excessive development of epithelial cells and of their derivatives filling the vesicles.

(*b*) Suppurative changes occur at times in these spots and give rise to abscesses, the specific nature of which, however, may still be considered doubtful, since similar processes also occur in the non-syphilitic forms of catarrhal pneumonia.

(*c*) Foerster, however, has in some of these cases met with a gradual thickening of the walls of the alveoli by the growth of a fibre tissue mingled with ovoid nuclei surrounding the spots of lobular pneumonia. These then become hard, smooth, pale and glistening, and in a later stage they show a yellow change which gradually extends throughout the nodule. This process has the greatest analogy with the growth of tubercular granulations, and if due to the syphilitic poison it would establish a close anatomical affinity between its effects and the changes which are most distinctive of tubercles. Similar appearances have been described, though on rather a larger scale, by Von Baerensprung and others, when the nodules so formed may attain the size of a walnut. Virchow has also remarked that these may co-exist with peribronchitic thickenings, and that they may pass in spots into ulceration; and he further observes, that when met with in stillborn children of syphilitic parentage, their specific nature is rendered the more probable from the fact that tubercle proper is never met with as a disease of the foetus.

(*d*) Virchow is also disposed to regard as being in some cases of syphilitic origin, indurated masses of fibrous structure more or less pigmented, and presenting a raspberry-like appearance, which are found scattered through the lungs. They are either seated immediately under the pleura, where they cause puckering and contraction, and also around the bronchi, where they form a cicatricial tissue, and they are often attended by pleural adhesions; cheesy spots are not uncommonly found scattered through them. The nature of these is however still more doubtful, since such masses are very common in the indurating form of tuberculosis when there is no suspicion of syphilis.[1] Virchow states that the more fibrous structures present no distinctive features of difference from the indurating forms of chronic pneu-

[1] Addison regarded these as pneumonia, and Virchow also speaks of them as the results of chronic pneumonia. For the reasons before given, I venture still to express an opinion respecting their tubercular nature. An appearance of this kind is described by Cornil as syphilitic (Bull. Soc. Anat. 1861).

monia which occur in the "grinder's asthma," and probably also in the whole class of diseases produced by the inhalation of irritating solid particles into the lungs.

(*e*) Virchow is further disposed to consider that fibrous induration of the pleura, and also certain forms of peribronchitic thickening which extend into the pulmonary tissue, may be due to the syphilitic dyscrasia, and that they may hold a place analogous to the cirrhotic indurations of the liver, and to indurations which are met with in the testicle under the same influence. Dr. Wilks has also raised the question whether some forms of "cirrhosis" of the lung may not have a similar origin, but this point still remains to be settled by further observation.[1]

(*f*) Virchow has also met with a change in the lungs closely analogous to the brown induration to be hereafter described, but occurring independently of heart diseases, and which from its associations he thinks may also be placed in this category.

(*g*) Dr. Hermann Weber,[2] in a case where there was evidence of constitutional syphilis, and where nodules which he was disposed to regard as early forms of gummata existed in the liver, found in the lungs a general enlargement of the superficial lymphatics, which were filled with a thickened cheesy lymph which could be expressed from their interior. These enlarged lymphatics presented on section the appearance of white spots scattered over the lungs: their contents presented granular corpuscles with multiple nuclei. The bronchial glands were also enlarged, softened, and crowded with cells exhibiting considerable activity of growth. Dr. Weber regarded it as doubtful whether the pathological condition of the pulmonary lymphatics or of the bronchial glands constituted the primary affection. The appearances described, as Dr. Weber himself considered, differed in many respects from those which have hitherto been regarded as syphilitic.

Syphilitic growths in the lungs certainly bear a closer resemblance to tuberculous formations than is presented by almost any other morbid change in this organ. It is useless at present to revive the former speculations which have been held with respect to the influence of syphilis on the production of tubercle. The question, however, may be looked at in another aspect, and it would appear to be a subject for inquiry, how far a pre-existing "tuberculous" or "scrofulous" constitution may aid in the development of these special local manifestations. Syphilis has long been known to exhibit its most virulent characters in patients of this diathesis, and it appears to be not

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[1] Wagner relates a case of the same kind. Vidal (Traité des Mal. Vén.) describes in a syphilitic patient a condition of fibrous induration surrounding the bronchi, and extending into the pulmonary tissue. It was chiefly limited to the lower lobes. The condition of the bronchi is not mentioned. Vidal notices the resemblance of the tissue to that produced by a chronic periostitis. Proof of the syphilitic nature of these is, however, wanting. Lancereaux (loc. cit. p. 424) considers that cicatricial contractions of the lung may also be due to this cause, but this must be regarded at present as being simply hypothetical.

[2] Path. Soc. Trans. xvii. (Two plates of the appearances in the lungs and liver.)

impossible that such a predisposition may render the lungs specially liable to suffer from the syphilitic affection, the characters of which may be partially modified by the tuberculous tendency. Tubercular changes are in many points of view so closely allied to the processes of inflammation that it has become increasingly difficult with further research to assign to them any specific character; but in the lungs at least, whether occurring in the form of granulations or of an infiltration, they are almost constantly attended by a fibro-nucleated growth of the alveolar wall, in which sometimes the fibrous and sometimes the nuclear element predominates. It would appear also by no means improbable, in the light of recent researches on the production of tuberculosis in the lower animals,[1] that various poisons, as well as simple irritants, may serve as the starting-points for tubercular changes in predisposed individuals. I would not, without much further personal experience than I possess on this subject, venture to affirm that syphilitic changes in the lung are identical with tubercle; but it is impossible to study the observations of those who have investigated both processes, and particularly the researches of Virchow, without being convinced of the close analogy between them; and it would appear to me that the conclusion that some of the changes thus described as syphilitic have a quasi-tubercular nature, is at least quite as likely to be correct as the converse, viz. that a large number of processes hitherto considered tubercular should be ascribed, when found in syphilitic patients, to the exclusively specific effect of this dyscrasia.

The clinical history of these changes is as yet an almost untrodden ground. The majority of the reputed syphilitic affections of the lungs have been observed in still-born children, or when found in adults have only been accidentally discovered on post-mortem examination. Lancereaux cites a few instances where pulmonary symptoms had been present before death. In one of these, quoted from Vidal, and where the chief change was peribronchitic induration, there were the physical signs of consolidation at the bases, associated with slight hæmoptysis, little cough, and no fever, but with a dyspnœa gradually increasing in intensity, and apparently proving, at last, one of the causes of death. The duration of the disease in this case, after pulmonary symptoms were first observed, extended over two years.

In another case under Lancereaux's own observation, and where the presumed gummata had formed cavities surrounded by much induration, the affection was limited to one lung, and the physical signs were those of induration with excavation; hæmoptysis, however, occurred also in this instance, and the sputa, at first scanty, became subsequently copious and fœtid; œdema of the legs, and slight pyrexia were present, and the patient died cachectic.

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[1] It is certainly a remarkable fact that in my experiments guinea-pigs inoculated with syphilitic virus were the only class that completely escaped secondary tuberculization; but this, when the difference of species is considered, would be no argument against the possible effects of this virus in the human subject. See lecture by the author " On the Artificial Production of Tubercle."

Lancereaux remarks that a unilateral affection of the lung, with signs of chronic induration or excavation, and in the presence of a syphilitic history, may lead to the diagnosis of its specific origin, but it must be remembered that the syphilitic affection is not invariably confined to a single lung.

In respect to treatment, Lancereaux cites several cases where a mercurial course has been followed by the cessation of phthisical symptoms, and by the improvement in some instances of the physical signs of the disease. I have more than once subjected phthisical patients with a history of syphilis, to treatment both by mercury and by iodide of potassium, but the results which I have hitherto obtained have been by no means favourable. The treatment by iodide of potassium would appear to be the least dangerous, and the most deserving of a more extensive trial.

BROWN INDURATION OF THE LUNG.

By Wilson Fox, M.D., F.R.C.P.

SYNONYMS.—Pigment Induration (Virchow); Brown Condensation (Zenker); Carnification Congestive (Isambert and Robin).

HISTORY.—This state of the pulmonary tissue occupies a doubtful ground between the indurations which succeed to long-continued congestion and the processes defined as chronic inflammation. Some of the difficulty of determining its exact nosological position depends on discrepancies in the statements made by different observers with respect to the exact changes which lungs to which this term has been applied have presented.

It was described originally by Andral[1] under the title of Hypertrophy of the Lung, and as existing in cases of chronic catarrh. He states that the change consists in an enlargement of the vesicles, together with thickening of their walls, and Rokitansky[2] appears to have observed a very similar condition. It has also been noticed by Hope[3] and Hasse.[4]

MORBID ANATOMY AND PATHOLOGY.—The state to which the term Brown Induration is applied is the result of long-continued congestion, most commonly arising either from marked incompetency or constriction of the mitral valve. Virchow,[5] who first after Andral gave a minutely detailed account of its appearances, also describes the lungs as enlarged, prominent, and not collapsing when the thorax is opened. They feel more compact than the normal lung, and they are also heavier and inelastic; they crepitate but little, and have a peculiar tint of yellow, shading into a brown or a reddish brown. On section the tissue is dense and is speckled with red spots of variable size, shading into blacker tints, and between these also the tissue has a more or less rusty appearance. A brownish fluid (brown œdema, Virchow) exudes on pressure. Virchow described the essential characteristic

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[1] Prec. Path. Anat. ii. 516.
[2] Path. Anat. 1861, iii. 46.   A drawing accompanies this description.
[3] Morbid Anatomy.
[4] Loc. cit. Hasse appears to have proposed the title of "Brown Induration," which seems, from its simplicity, to be the most eligible for this affection.
[5] Archiv, i. 1847, p. 461 et seq.

of this condition as depending on the accumulation of hæmatoidine in the epithelial cells of the air-vesicles, which are either natural or more or less enlarged, and also in granule cells, which probably result from the transformation of the former. The pigment is for the most part in the form of granules, insoluble in acetic acid, but which are destroyed by caustic alkalies and by sulphuric acid. Various transformations of the yellow pigment into black granules can be seen within the cells themselves, and later it is found free in the walls of the alveoli and in the interstitial tissue. Further accounts of this state have been given by Friedreich [1] and by Buhl.[2] The latter describes and figures a series of varicose dilatations of the capillaries co-existing with the pigment in the walls of the alveoli. Friedreich's description of the filling of the alveoli with enlarged epithelial cells, and with the products of their proliferation, agrees very closely with Virchow's.

The point on which most difference of opinion exists is that which refers to the thickening of the alveolar walls. Virchow does not describe this change, and Zenker [3] says that he has not met with it. Rokitansky, however, figures it, and Isambert and Robin,[4] who, under the title of "Carnification Congestive," have described a very similar condition, state that the walls of the alveoli and the interstitial tissue, in addition to containing a large quantity of pigment, are infiltrated with an amorphous exudation matter. In the specimens which I have examined I have found such thickenings in considerable tracts, together with a distinct increase of fibrous tissue in the walls of the alveoli; but this change is not uniformly present, and in other places the alveoli are found filled with catarrhal cells, while their walls present no other change than that arising from the distension of the capillaries. I have also observed a considerable thickening of the coats of the branches, both of the pulmonary artery and of the pulmonary vein; an appearance, however, which has not been described by other writers.

The change in the lung has appeared to me to be referable to two stages. In the first there is intense congestion, sometimes general, but more commonly found in limited parts, and in these congested parts a considerable amount of pigment may be seen in the pulmonary epithelium. Such parts float in water, and are more or less œdematous, yet crepitant, but comparatively inelastic. Their tint is of a uniform reddish brown. In the later stages the pulmonary alveoli gradually become filled more or less completely with epithelial products resembling those of catarrhal pneumonia, and the tissue, to a great extent, loses its crepitant character. In this stage also it is not so prominent, and closely resembles a congested and collapsed lung, except that the surface is finely granular, and is mottled with spots of

[1] Virchow, Arch. x. 201. Friedreich describes corpora amylacea as existing in such lungs. I have also seen these under similar circumstances.
[2] Ib. xvi. 559. Drawings accompany this description.
[3] Beitrage zur Normalen und Path. Anat. der Lungen.
[4] Mém. Soc. Biol. 1855, 2ᵉ Sér. ii. p. 3 et seq.

yellowish pigment on the brown and indurated tissue. It has n
the friability of ordinary pneumonia, but is comparatively toug
inelastic. This latter change corresponds with the "carnifi
congestive" of Isambert and Robin, and the parts so affected s
water, but not in all portions. The alveoli are loaded with epi
cells and with granule cells containing an excessive amount of
toidine. Isambert and Robin describe the pigment as som
existing in the crystalline form, and this, in the condition of me
can be seen in the walls of the alveoli and in the fibrous tiss
rounding the arteries and veins.

The extent of lung thus implicated varies considerably. The
may exist only in patches, or it may extend to considerable t
tissue. I have seen it throughout the greater part of the low
Isambert and Robin have seen it affecting the whole of one a
greater part of the opposite lung.

The appearances thus presented are quite different from t
hæmorrhagic infarcta, though these are not unfrequently pre
other parts of the lung. The parts affected want both the dens
also the prominence of portions of lung into which hæmorrh
occurred, and the escape of the colouring matter of the blood
to be due to mere capillary rupture, and not to any extensive
vasation. The nature of the change seems to depend on lo
tinued congestion gradually giving rise to a catarrhal pneumon
chronic type, and the thickening of the alveolar walls may pr
occur in the later stages of this process,—thus creating an
with some of the other forms previously described.[1] Zenker
that this pneumonia may pass into true hepatization, though fi
exudations are commonly wanting. The pneumonia, howeve
at times be mingled with so much extravasation as to giv
hæmorrhagic character.

The enlargement of the lungs described by Virchow has
peared to me to be essential to the process—at least such enlar
has not existed in two of the best marked instances of the
which have come under my own observation. It would appe
improbable that, when such enlargement has existed, the lun
been affected by emphysema during or prior to the other c
Zenker states that an extreme degree of atrophous emphysema
in some specimens which he examined.

This change, according to Zenker, appears to be more commor
than after the age of forty.

SYMPTOMS.—Bamberger[2] describes the earlier conditions
state as associated with diminished resonance on percussion, to

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[1] Grisolle, p. 71, describes two cases of chronic pneumonia associated with hea
tion, but the appearances observed do not show positively that they belonged
class. In one the condition was simply that of grey induration; in the o
tissue was of a reddish tint and finely granular, but in other parts it presented
appearance of being of recent origin.
[2] Lehrb. Krank. der Herzens, 204.

ith weakened respiratory murmur; but these physical signs are
ommon to many cases of pulmonary congestion from cardiac disease
hen the induration now described does not exist.

Dyspnœa is commonly present, and cyanosis is observed in extreme
ases; but neither these nor the rusty sputa often seen are necessary
igns of the condition in question.

Isambert and Robin describe dulness on percussion, together with
ronchial breathing over the affected parts, and this also has existed
a one case which I have observed.

The temperature in both the best marked cases which have come
nder my observation has been elevated, but not exceeding 102° Fahr.
a one case a fluctuating pyrexia, sometimes reaching 102°, and
a other days not exceeding 99½° or 100°, continued during
arly a month—death finally taking place from gradually increasing
thenia and cyanosis: the heart was much hypertrophied, adherent
the pericardium, and presented extensive disease of the mitral valve.
nother case was complicated by erysipelas of the leg passing into
ngrene. It must therefore be regarded as doubtful whether the
rexia depended on the pulmonary condition. Rusty and blood-
ained sputa are common, but neither these nor any of the physical
gns as yet observed afford any positive grounds for the diagnosis of
is affection, though there might be strong reason to suspect its ex-
ence from the persistence of signs of consolidation during a long
riod, and associated with cyanosis and dyspnœa depending on
arked disease of the mitral valve.

The TREATMENT must be mainly directed to the cardiac conditions
esent. The indications for the relief of pulmonary congestion, such
the application of revulsives and counter-irritants, and the internal
lministration of stimulants, are those which would appear to be the
ost suited to this state. (See Secondary Pneumonias, p. 736.)

CIRRHOSIS OF THE LUNG.[1]

By H. Charlton Bastian, M.A., M.D., F

NATURE AND HISTORY.—This is a rare disease, mos
type, in which the individual has suffered, perhaps
from cough and muco-purulent expectoration, w
hæmoptysis; in which the wasting is not very mar
constitutional symptoms of the ordinary form of pht
absent. There is usually marked dulness, accompanie
and retraction, of one side of the chest, with or wit
sounds on auscultation; whilst there is generally incr
accompanied by puerile respiration, on the opposite s
is more or less displaced towards the affected side; w
be signs of dilatation and hypertrophy of its right cav
with anasarca and ascites. After death the lung on tl
is found to have become shrivelled to one-half or eve
its natural size—owing to its conversion into a tough
with obliteration of its air-cells and usually more or
of its bronchi; whilst that on the opposite side is
and presents no evidence of the existence of tube
disease.

This pathological condition was incidentally alluded
as a variety of dilatation of the bronchial tubes, and
referred to by Dr. C. J. B. Williams, only a few w
appearance in 1838 of a most interesting memoir on
Sir Dominic Corrigan.[3] In this memoir the disease
described, so far as the state of knowledge at the tir
an entirely new interpretation was given of its patholc
name was proposed, on account of the close resem

applicable as it can well be to the lung affection about to be described.[1]

Whilst Laennec, in his admirable account of dilatation of the bronchi—a morbid state which had never been previously described—looked upon the condensation of tissue around the dilated tubes as being invariably secondary to and the effect of the dilatation, Corrigan, on the other hand, maintained that in a certain number of cases, which he proposed to range under the name "Cirrhosis of the Lung," the fibroid metamorphosis and induration was the primary and essential anatomical lesion, and that the dilatation of the bronchi was only a secondary effect. Omitting for the present the consideration of the question as to whether Corrigan was correct in the explanation he offered of the mode of origin of the bronchiectasis, I may state that his main position appears to have been a correct one. It seems to be doubtedly true that, in a certain number of cases in which dilated bronchi have been met with after death, an original fibroid conversion and shrinking of the lung-tissue has entailed this as a consequence: the bronchiectasis has been secondary, and not primary.

Notwithstanding the enunciation of Corrigan's views, however, the French pathologists, with the exception of M. Jaccoud, adhere to Laennec's interpretation of the sequence of these phenomena; and Cirrhosis of the Lung is, moreover, scarcely considered to be entitled to rank as a distinct disease by many English and German pathologists. From facts subsequently to be mentioned, it will be seen that Corrigan placed too much stress upon the dilatation of the bronchial tubes. This is not an essential element in the disease, but is, rather, a very frequent accompaniment. It will be observed that in several recorded cases bronchiectasis was either absent altogether or only very slightly marked. In these cases the fibroid infiltration and shrinking of the lung, which are the essential characters of Cirrhosis, existed alone. Those who still doubt the propriety of regarding this as a disease with clinical characters of its own, distinguishable from bronchiectasis, may perhaps be influenced by an attentive consideration of the following facts.

From the analysis of 43 cases by M. Barth,[2] and by Lebert[3] of 24 cases of bronchiectasis, it appears that this affection most notably increases in frequency with advancing age, and that by far the larger proportion of cases are met with in persons who are more than 60 years old. Thus in Lebert's 24 cases, it was met with four times before the 10th year, ten times from the 10th to the 55th year, and ten times

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The name "Cirrhosis" was, in fact, originally given by Laennec to the now well-known liver disease, under the influence of a misconception as to its nature. He thought that it was due to the deposition within the organ of a peculiar morbid substance of a tawny or rust-brown colour. These patches and islets, however, are now known to be for the natural acini of the liver, bile-stained and isolated by what is the real anatomical element of the disease—the new growth of fibre tissue.

Rech. sur la Dilatat. des Bronches, Mém. de la Soc. Méd. d'Observat. de Paris, tome iii. (1856), p. 469.

Anat. Patholog. tome i. p. 620.

from the 55th to the 85th year ; whilst according to Barth it was
with as follows :—

No. of Cases.									Age.
2	.	.	.	.	.	.	.	.	1 to 20th year.
3	.	.	.	.	.	.	.	.	20 — 30 ,,
3	.	.	.	.	.	.	.	.	30 — 40 ,,
4	.	.	.	.	.	.	.	.	40 — 50 ,,
5	.	.	.	.	.	.	.	.	50 — 60 ,,
7	.	.	.	.	.	.	.	.	60 — 70 ,,
19	.	.	.	.	.	.	.	beyond the 70th year.

But an analysis of 30 cases of Cirrhosis of the Lung, which I
collected, appears to show a most striking difference as regards
prevailing age at which this lung affection is met with in the p
mortem room, and that at which dilatation of the bronchi is
countered.[1] Thus, in the 30 instances of Cirrhosis the following
were attained :—

No. of Cases.								Age.
2	.	.	.	.	.	.	.	1 to 15th year.
3	.	.	.	.	.	.	.	15 — 20 ,,
7	.	.	.	.	.	.	.	20 — 30 ,,
9	.	.	.	.	.	.	.	30 — 40 ,,
2	.	.	.	.	.	.	.	40 — 50 ,,
3	.	.	.	.	.	.	.	50 — 60 ,,
4	.	.	.	.	.	.	.	60 — 70 ,,

From these figures it appears that 19, or almost two-thirds of
total number of cases of Cirrhosis, occurred between the ages of
and 40 ; whilst of Barth's 43 cases of bronchiectasis, only 7, or
than one-sixth of the total number, were met with at the same
On the other hand, more than one-half the cases of bronchiect
(26 : 43) were in individuals over 60 years of age ; whilst rather
than one-seventh (4 : 30) of the cases of Cirrhosis were encount
after the same year. Even these facts alone tend strongly against
view that well-marked fibroid infiltration with shrinking of the
is to be considered as a sort of sequence of dilated bronchi. Whils
the other hand, seeing that bronchiectasis is met with in such a l
proportion of the cases of Cirrhosis of the Lung occurring at the
above mentioned—when dilatation of the bronchial tubes is other
very rare—there are strong grounds for the opinion that such a co
tion of the lung is especially favourable to the production of mo
less dilatation of the bronchi. Other striking differences, how
exist between the two affections. Thus, well-marked Cirrhosis is al
invariably confined to one lung : not so with bronchiectasis.
or less hæmoptysis was present in more than one-half (17 : 30) of
cases of Cirrhosis, in only four of which was anything which o
be called " tubercle " said to be present in one or other of the lun

[1] Although it is perfectly true that Barth's cases (with one exception) were coll
at the general hospitals for adults, and also at the Salpêtrière, and therefore ma
at all fairly represent the frequency of the disease in childhood ; still, that his figur
show the determining influence of age may be also seen from the fact that in the c
of six years 25 examples were met with at the Salpêtrière, whilst only 18 were met
during 25 years at the general hospitals of Paris.

[2] In only one of these four cases did the " tubercle " exist in the cirrhosed lung.

whilst the same symptom was met with in less than one-sixth of Barth's cases of dilated bronchi (7 : 43)—and of these no less than four were also suffering from phthisis. There are differences, more-over, as regards sex. According to Lebert,[1] dilatation of the bronchi is as common in females as in males; whilst only one-fifth of the total number of cases of Cirrhosis have been observed in females.

Though believing that in the majority of cases condensation of the lung-tissue from fibroid metamorphosis precedes the dilatation of the bronchi with which it is so often associated, still it would appear quite obvious that in some other cases the order is just the reverse. What Laennec maintained to be the rule, does really obtain in some cases: the cirrhotic change is then secondary to the bronchiectasis. The two morbid conditions have undoubtedly most intimate relations with one another, and occasionally it may be difficult to pronounce which was the primary lesion. There is no reason, of course, why the cirrhotic change should not invade a lung whose bronchi are dilated, just as it invades one in which the bronchi are healthy. But in the cases where this has been the order of events, the amount of condensation and induration of lung-tissue is far greater than what is often entailed by the mere dilatation of the bronchi. So that, although there may have been some amount of pre-existing bronchiectasis, Cirrhosis afterwards becomes the predominant affection.

But there is another condition of the lung, known for the most part by the name "chronic pneumonia," under which are recorded cases that may better be regarded as instances of Cirrhosis of the Lung. Much uncertainty and confusion have resulted from the use of the former term, on account of the shifting signification which has been given to it by different writers. But the perusal of Charcot's memoir, " De la Pneumonie Chronique,"[2] and of the account given by Grisolle in the last edition of his work " De la Pneumonie," cannot fail to convince the reader that, instead of referring to any condition of lung especially characterised by the impaction of the air-vesicles with a more or less solid accumulation of cells or cellular débris, these writers understand this name to imply a fibroid infiltration of more or less of the organ, and the gradual substitution of a tissue of this kind in the place of the proper substance of the lung.

Pathological states of the lung very similar to, or even identical with this, were originally described by Laennec[3] as forms of infiltrated " tubercle," under the names of *grey tubercular infiltration* and *jelly-like infiltration.* The tubercular nature of these morbid states was afterwards denied by Chomel,[4] who looked upon them as evidences of a non-specific chronic pneumonia—a view which has been more or less adopted since his time by succeeding pathologists. Andral[5] described

[1] Barth does not give the proportion of males to females in his observations.
[2] Paris : Thèse, 1860. Containing copious references.
[3] Diseases of Chest, translated by Forbes, 4th Edit. 1834, p. 256.
[4] Art. " Pneumonie Chronique," Dict. in 25 vols., 1842.
[5] Précis d'Anat. Patholog. tome iii. p. 517 and Clinique Médicale.

a *red*, a *yellow*, a *grey*, and a *melanic induration* of the lung, which seems to represent only different stages and varieties of a fibroid infiltration of the organ, and correspond with what Hasse,[1] Rokitansky,[2] Förster,[3] and other German pathologists mean by *interstitial pneumonia* and *lungen-induration*. Lebert[4] also described a *hepatisation indurée*, and a *hepatisation jaune;* whilst Cruveilhier,[5] referring to the later stages of the same pathological transformation, spoke of a *phlegmasie indurée* and an *induration mélanique ardoisée*, understanding that the essence of these conditions was a "métamorphose fibreuse" of the proper lung-tissue. Addison[6] also described two of the sequences of acute pneumonia under the names *albuminoid induration* and *iron-grey induration*. With regard to the second of these pathological states, this undoubtedly corresponds with the fibroid induration of other writers, and a careful examination of Addison's plates, together with a comparison of the descriptions given of the two conditions, almost suffices to show that the first is but a rarer modification of the second pathological state—into which it often seems to pass by insensible gradations. Dr. Wilks[7] also describes chronic pneumonia as a fibroid induration of the lung substance, due to an actual new growth of fibre-tissue which slowly increases in amount.

Thus there seems to be a pretty general agreement between the writers I have named (some of the principal of those who have written upon the subject), concerning the essential nature of the condition which often goes by the name "chronic pneumonia." When affecting any considerable extent of the lung it has been generally recognised as a condition of great rarity. Charcot imagined that at the time he wrote there were only about ten or twelve cases on record, which could be indubitably regarded as examples of this disease. Grisolle, also, had only met with six cases during his long experience. Both these writers believe that this state of the lung may be the almost immediate sequence of an ordinary acute pneumonia, although they think that at other times it is chronic from the first, and commences in the most obscure and insidious manner. Both these writers also, as well as most of the others I have mentioned, are fully satisfied that the minute anatomical characters of this so-called chronic pneumonia are essentially similar to that of the ordinary indurated tissue surrounding vomicæ or foreign bodies in the lungs. I need only add that the tissue-changes in these cases are essentially similar to those which Dr. Sutton has described as "fibroid degeneration of the lung," and that such a change, prevailing to a wide extent, is the anatomical characteristic of Cirrhosis of the Lung.

It seems to me expedient to do away altogether with the name "chronic pneumonia," as an appellation for the pathological changes

[1] Patholog. Anatomy (Syd. Soc. Translation), 1846.
[2] Man. of Path. Anat. vol. iv. (Syd. Soc. Translation), p. 60.
[3] Lehrb. der Patholog. Anatom. p. 296. Jena, 1862.
[4] Anat. Patholog. tome i. p. 648.
[5] Ibid., livraison xxxii. p. 8 ; and Anat. Patholog. Génér. tome iii. p. 608.
[6] Guy's Hosp. Reports, 1843, p. 365.   [7] Lect. on Path. Anat. 1859, p. 256.

in question. This seems desirable for the following reasons :—1. Any pathological state to which the term "chronic pneumonia" is applied ought to be characterised by anatomical characters similar in kind to those which are met with in the acute condition—conditions which are fulfilled by the "chronic lobular pneumonias" of phthisical patients. 2. Admitting that the fibroid overgrowth and substitution, which has been hitherto styled "chronic pneumonia," is sometimes the direct sequence of an acute pneumonia, still this secondary condition is not a modified persistence of the old state, but is due to the supervention of an entirely new and different process: in these cases, in fact, we have to do with a sequence to, rather than with a chronic persistence of, the original malady. 3. Although such a pathological state is occasionally the direct sequence of an acute inflammatory condition, still in the large majority of cases it seems to be due to an essentially chronic process—to one which is deficient in some of the most important characters of an inflammatory change, and which more closely resembles a mere infiltrating new growth.

The term "interstitial pneumonia" seems to be almost as unsuitable as that of "chronic pneumonia," as an appellation for the fibroid indurations in question. Whether such changes are met with in the lung, in the liver, or in any other organ, their mode of initiation, progress, and minute anatomical characters, seem to be essentially similar. They advance insidiously, in the great majority of cases, without affording the least clinical evidence that the patient is suffering from an inflammatory disease;[1] and when the organs in which such changes had been advancing are submitted to microscopical examination, there is a similar absence of the signs of an inflammatory process. A new growth is met with, supplanting the proper anatomical elements of the part, and it seems to me to be no more suitable to speak of such a process as an inflammation than it would be to apply the same term to a slowly increasing but infiltrating cancerous growth.

The more partial and local changes might simply be styled "fibroid indurations," reserving the term "Cirrhosis" for the more extensive and advanced change, when it affects either an entire lung or at least one lobe of the organ.

From what has been said it will be seen how intimately related Cirrhosis of the Lung is, not only to bronchiectasis, but also to what has been hitherto called "chronic pneumonia." It will not be so much a matter of surprise, therefore, that some of the cases of which I have given an abstract in this paper, have been originally recorded under one or other of these names. No sharp lines of demarcation can exist between fibroid indurations of the lung ("chronic pneumonia" of other writers) and Cirrhosis, because they are merely

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[1] Dr. Wilks says (loc. cit. p. 237): "For my own part I believe such a process is essentially chronic, and at no time, if an opportunity had been given for examining such a lung, would it have presented any different appearances, except in amount ; growing indeed like a tumour, and, like it, having, no doubt, some elementary forms preceding the fibrous structure, but the mode of production and development so slow and continuous that no distinct stages or changes in the structure can even be distinguished."

different degrees of one and the same pathological condition. Therefore, one or two of the cases that I have included amongst the thirty instances of the disease on which this paper is based, may seem doubtfully entitled to the latter name; but I have placed them in this series precisely because they serve to indicate this relationship, and to show what are the early stages of the disease which we are now describing.

Since diseases have no distinct and independent existence, but are merely groups of symptoms, or of pathological changes, which tend to repeat themselves with varying degrees of frequency, it is only to be expected that intermediate conditions should at times present themselves. Our nomenclature and classification of these sets of symptoms, or pathological changes, must inevitably be more definite and sharply defined than actual facts or occurrences would warrant. We can but seize upon certain combinations of symptoms or changes which are apt to recur, and ticket them in their typical condition, as so many "diseases;" though in doing this we should ever recollect that the symptoms or changes are not distinct and independent, but are variously related to, and miscible with, other possible combinations. With the distinct understanding that the diseases enumerated in our nosologies vary immensely, not only in respect to the frequency, but also in respect to the definiteness of character, with which they tend to recur; still, one must regard all such described diseases as little better than rallying points, round which special groups of symptoms or changes may be conveniently ranged.

The disease which we are now considering is comparatively rare, and it can only be arbitrarily marked off from the fibroid indurations of smaller extent, out of which it is developed. Still, a certain set of symptoms do tend to recur in association with a certain set of advanced anatomical changes, and these have been ticketed as a disease which is distinguished by characters of its own, as much clinical as anatomical. On these grounds Cirrhosis of the Lung has the same right to be considered as a distinct disease, that many others possess whose claim to a place in our nosologies is unquestioned.

This paper is essentially based upon an analysis of thirty recorded cases of the disease.

One of these cases was originally reported by Sir Dominic Corrigan; one has been taken from Dr. Sutton's paper; and two others are from M. Charcot's memoir, "De la Pneumonie Chronique." On the other hand, seven cases have been included which had been described under the head of Bronchiectasis.[1] One of these was recorded by Laennec (though quoted by Corrigan as an instance of Cirrhosis); one was observed by Dr. Stokes; one by Dr. Bright and Dr. Hughes;[2] two are from M. Barth's memoir; whilst the last of the cases previously recorded under the head of Bronchiectasis, has been

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[1] These are here recorded, because they not only serve to show the intimate and natural relationship existing between the two "diseases," but also because, owing to the extent and character of the morbid changes met with, they have almost an equal title to be ranked under either head.

[2] See Guy's Hospital Museum, with description in catalogue.

taken from MM. Hérard and Cornil's recent treatise.[1]   The remaining nineteen cases of which I have given abstracts were recognised as cases of Cirrhosis of the Lung, and sixteen have been published as such—fifteen in one or other of the periodical publications of Great Britain and Ireland, and one in Paris during the present year (1867)[2] by M. Jaccoud.   The other three cases have not been hitherto published : one occurred in the practice of Dr. Gull at Guy's Hospital, and one in that of Dr. Pollock at the Brompton Hospital, and to each of these gentlemen I have to express my best thanks for their kindness in placing reports at my disposal.   To Dr. Wilks I am also much indebted for granting me access to, with permission to publish, the records of a case ·which formerly occurred in the practice of Dr. Addison, at Guy's Hospital.

In order to show the kind and range of variation met with in different cases, I have deemed it most advisable to give a tabular abstract of these cases, which it is hoped will be of use for future reference.   (See p. 837.)

PATHOLOGICAL ANATOMY. — Adhesions of the pleural surfaces, serving to unite the affected lung to the parietes of the chest and to the diaphragm, have been met with in almost every case.   They were reported as present in twenty-six out of the thirty cases ; only in one case (I.) were they stated not to exist, whilst in the remaining three the presence or absence of adhesions was not noted.   Of the twenty-six cases in which the adhesions are described as existing, they were somewhat loose in five, but firm, tough, and often even cartilaginous in consistence in the twenty-two other cases.   In nine of these the adhesions were more or less partial, whilst in thirteen, or nearly one-half of the total number, they were general, and the lung was at the same time usually much reduced in size.   Adhesions between the diseased lung and the pericardium were not uncommon, and in one case the posterior surface of the greatly enlarged opposite lung was also united to the diseased organ.   Where the adhesions were well developed and general, it was frequently necessary to cut the lung out of the corresponding side of the thorax ; and more or less extensive plates of firm fibro-cartilaginous-looking material were found covering a certain portion of the surface of the organ, and gradually shading away peripherally into an ordinary tough fibrous coating.   This layer over certain parts of the lung, having a fibro-cartilaginous appearance, may be more than an inch in thickness—and then its inner strata evidently correspond in situation to what had previously been proper lung-tissue.   In only one case (XXII.) was any fluid found, and in this the pleura is stated to have been nearly one inch in thickness ; whilst in a cavity between the adhesions, which were only partial, there was contained nearly a quart of clear serum.   The lung was, moreover, only

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[1] De la Phthisie Pulmonaire, Paris, 1867.

[2] It seems only right to state that this paper has been written nearly three years and a half—ever since October 1867.   A few other cases have been recorded since this date.

as large as a man's fist; its tissue was remarkably hard, and its bronchi were not dilated.

The size of the lung, in the more recent cases, has undergone no appreciable alteration; in all the more chronic cases, however, it has exhibited a variable amount of shrinking. This is often very considerable: in one remarkable case (XXIII.) it was scarcely the size of a man's hand, and there was no pleural effusion of any kind (to help to bring about the contraction), similar to what existed in the other case (XXII.), in which the size of the lung was reduced to such an extreme degree. All intermediate grades are to be met with between this amount of contraction and the normal dimensions of the organ. On section, it is often seen that the lobes of the lung are firmly connected together by a dense fibro-cartilaginous material, similar to what more frequently occurs in connection with the pleura on the surface of the lung. The tissue of the organ varies much in appearance in different cases, owing to the different degrees of progress which the disease may have made; and also to the varying amount of black pigment present, and to the number and mode of distribution of dilated bronchi or ulcerated caverns throughout its substance. Occasionally, islets of healthy lung-tissue are left here and there, in the midst of the fibroid induration. The disease may affect only one lobe (XV.), the two lobes unequally, or the whole organ pretty uniformly. When existing in its early stages, either generally or partially (III., XIV.), the nature of the pathological change is even then most obvious to the naked eye. The texture of the lung being firm, tough, dense, and incapable of being broken down by the finger, one sees a smooth or only very slightly granular surface of a blackish or iron-grey colour, intersected in all directions by white bands of ligamentous-looking tissue, often forming a sort of trabecular network, and dotted with white circles of varying sizes, produced by the cut walls of the thickened smaller bronchial tubes. Very often, in its early stages, this invasion of fibre-tissue is most distinctly seen to extend inwards from a greatly thickened pleura. For continuous with it may be seen portions of lung-tissue which have been completely converted into a fibro-cartilaginous-looking material; whilst this may pass, internally, into a simple ligamentous-looking texture. Still further internally there is a trabecular structure, such as I have just described, the white bands of which become narrower and narrower, and may gradually fade away into almost unaltered lung-tissue. In other cases where the consolidation spreads from different centres within, rather than from the surface of the organ, the fibroid thickening and white bands seem to radiate principally from the thickened walls of the bronchi. In its more advanced form, almost the whole organ, or large parts of it, grate under the knife when a section is made, cutting more like a tendon or mass of fibro-cartilage than anything approaching to normal lung-tissue, and whole tracts of it may in this later stage present the smooth yellowish-white appearance of cut tendon, and be almost free from pigment (IV., XXVII.) As the fibroid induration

advances, air-cells and vessels become more and more obliterated; the lung-tissue gradually yields less and less fluid when squeezed, and becomes at the same time more incompressible.

In the great majority of instances, changes such as I have mentioned are those which are apparent from the very commencement of cases of Cirrhosis of the Lung. But on those rarer occasions when the cirrhotic process is the direct sequence of an acute pneumonia, the first process is one which has been termed *induration rouge* by Andral and other writers. We have an instance of this change in the case (II.) recorded by Dr. Sutton,[1] when, on section, the upper lobe of the right lung was of a *dark red* colour, and the interlobular tissue appeared to have undergone an increase. Only a very small quantity of fluid appeared on the divided surface, and the tissue did not easily break down under the finger. The whole of the lower half of this lung was solid, firm, and somewhat tough. It had a *reddish-grey* colour, and offered some amount of resistance to the knife; whilst it sank in water, and exuded scarcely any fluid on pressure. Here the lower lobe was evidently in a more advanced stage of the disease than the upper, and it seems to have been in much the same condition as the upper lobe of the left lung in the case (V.) of M. Legendre. A more advanced stage is recorded in the case of the child (I.) reported by Sir D. Corrigan, where the right lung was solid, non-crepitant, *greyish-red*, *tough*, and traversed in all directions by thickened white bands of fibro-cellular tissue—though there still seemed to have been no contraction of the affected organ. But in the case (III.) of the man observed by M. Charcot, who had suffered from an attack of acute pneumonia about four months before his death, the disease seems to have made rapid strides, and presents us with a still more advanced phase. The whole right lung was pretty equally affected, and had undergone an evident diminution in volume.[2] Its tissue was so dense that the finger could not penetrate it, and, on section, it resisted the scalpel like fibro-cartilage. The three lobes were seen to be firmly united, and the surface of the section was smooth, non-granular, *greyish-blue marbled with black,* whilst pale ligamentous partitions of fibrous tissue subdivided the lung in all directions, and formed a minute network.

These are the stages by which the Cirrhosis that supervenes as the sequence of an acute pneumonia appears gradually to approximate to the condition of the lung which is characteristic of the earlier stages of the more chronic process.

In only one out of the thirty cases which I have tabulated is there

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[1] It seems to me most probable that this state of the lung was the sequence of an acute pneumonia. This must either have been the case, or else it must have been due to the supervention of an acute fibroid change without the existence of a previous pneumonia. Even if the latter alternative were true, the results would seem to be much the same in either case, since the condition of this lung appears to agree in all respects with Andral's description.

[2] It was one-third smaller than the right lung, which was noted as being very large. The actual amount of contraction of the cirrhosed lung, therefore, had not been very great.

any certain evidence of the existence of even a small quantity of "tubercle" in the cirrhosed lung. This was in the case (II.) of M. Jaccoud, when a very small quantity of crude and slightly softened[1] "tubercle" was found in the posterior part of the apex of the lung affected. In three other cases, however (V., IX., XII.), a small amount of "tubercle" was said to have been found in the non-cirrhosed lung.

But, although the existence of "tubercle" in cases of Cirrhosis seems to be a perfectly accidental occurrence, the same cannot be said with regard to the presence of *ulcerated caverns* in the indurated lung-substance, since these have been met with in about one-fourth of the total number of cases. Sometimes these caverns appeared to have been formed slowly, owing to the molecular disintegration of portions of the new tissue which had undergone a fatty metamorphosis, whilst at other times they have originated by a gangrenous process, as occurred in one (XV.) of M. Charcot's cases. Here, one of the excavations had irregular walls, and seemed to have arisen by a gangrenous process about two months previous to the patient's death; whilst another appeared to have been on the eve of forming, and was represented by a softened patch of yellowish tissue, with a disagreeable, though not gangrenous odour. In a case reported by Dr. Mayne (VII.), the patient died from the supervention of gangrene in the diseased lung, though there were no caverns. Towards the lower part of the consolidated organ the tissue had the olive or purple tint of gangrene with a corresponding odour. In all the recorded cases, however, in which caverns existed in the lung, save the one previously mentioned, they seem to have been formed by the slower process of ulceration or molecular disintegration, since there was no preceding history of gangrene. In two cases (IX., XVII.) a recent coagulum of blood was found in the ulcerated cavity; in one (XXIII.) the cavity was old and very large, being 4″ in length by 2¾″ in breadth; in two (XIII. and XVIII.) the cavities were single and small; in one (XXX.) there was ulceration of portions of the walls of two bronchial dilatations; whilst in another (XXIV.) case there was a small excavation of the size of a hazel-nut, whose nature was doubtful. In only one of these cases were there several excavations existing in the same lung, and that was in Case XVII.

In addition to these ulcerated cavities—having more or less ragged walls, and bounded by lung-tissue rather than by an altered mucous membrane—there are usually found other cavities and enlarged canals resulting from *dilatations of the bronchi*. These are not commonly met with in the early stages of the disease, such as have been hitherto spoken of under the name of "chronic pneumonia," and they are by no means always present, even when the disease is fully established and when great contraction of the lung has taken place.

There was no dilatation of the bronchi at all in one-fifth[2] of the

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[1] Even this was, therefore, in all probability, merely a cheesy patch of chronic lobular pneumonia.

[2] Nos. II., III., VI., X., XV., XXII.

thirty cases which I have collected, and in four other cases[1] it was present only to a very slight extent. In one-third of the cases, therefore, it has been either altogether absent, or else an insignificant feature of the disease. In the remaining two-thirds of the cases it existed to a variable extent. In one of the cases—that of a child— where the amount of dilatation was extreme (V.), and in which, moreover, the fibroid change seems to have advanced upon a lung whose bronchi were already dilated, it was a most typical instance of what has been called *uniform* dilatation. The bronchi were found to be healthy as far as their first division, but beyond this point, instead of diminishing at the successive bifurcations, they preserved the same calibre as far as their termination—and in some places the diameter of a distal branch was even greater than that from which it proceeded. At their extremities there was a simple *cul de sac*, and no tendency towards the formation of an ampulla. The mucous membrane was greyish-black,[2] slightly villous, and evidently thickened. In two other cases recorded by Barth (XXIX., XXX.) and occurring in adults, in which the amount of dilatation was extreme, it was of the *mixed* kind —consisting partly of cylindrical and partly of spheroidal dilatations. But in these two cases also it seems most probable, from a consideration of the histories of the patients, that dilatation of the bronchi had existed for many years before the fibroid change made any notable advance in the diseased organs.[3] The mucous membrane lining the variously dilated bronchi was in both cases smooth, dark red, and thickened. In only one other case (VIII.) was the amount of bronchial dilatation extreme. Here the lower lobe of the affected lung contained an extensive series of bronchial cavities between the size of a fowl's egg and that of a sparrow, some of which were partly filled by a semi-solid mucous secretion. In Case XXVI. one of the cavities was as large as an apple, and in No. XXVII. two large cavities—each as large as an egg—were the only ones existing. In other instances the dilatations were much smaller; thus in Case VII. a vast number of little cavities existed, varying in size between that of a pea and a marble, and all full of a muco-purulent secretion. In other cases caverns, varying in number, and of all sizes between these extremes, were encountered. The more or less spheroidal cavities were almost invariably associated, also, with cylindrical dilatations of the tubes; and in some cases the rounded enlargements were decidedly more common towards the periphery of the organ. The condition of the membrane lining the dilated bronchi has only been specified in twelve cases: in seven of these it was dark red, congested and thickened (and in two of them even velvety or slightly

[1] Nos. XIII., XX., XXIII., XXV.
[2] Most probably a post-mortem coloration.
[3] These cases, in fact, seem to me to belong just as much to the subject of "Bronchiectasis" (under which they were described by Barth) as to that of "Cirrhosis." I have included them here simply because they serve to show the intimate and natural relationship that occasionally exists between the two diseases.

villous), whilst in the five others it was rather a smooth, dull, or glistening membrane. In none of the cases is there any mention made of the slightly prominent transverse striæ which are so often met with in dilated bronchi according to Barth, and which I have myself seen extremely well developed in one instance, where the mucous membrane covering an enormous extent of dilated bronchi had quite a reticulated aspect, owing to the thickening of transverse and longitudinal fibres external to the mucous coat.[1] The bronchial dilatations are occasionally empty, though they are generally found to contain a considerable quantity of pus or muco-pus,—this being often thin, but at other times thick, tenacious, or even semi-solid in consistence, owing to partial inspissation. This fluid may be blood-stained,'and it has often a peculiarly stale, disagreeable odour, amounting in some instances even to foetidity.

In two cases (XXVI., XXVII.) there were emphysematous bulla observed on some parts of the surface of the diseased lung : in one they were situated on the upper lobe, and in the other, dilatations the size of a nut skirted the anterior border of the lower lobe.

Modifications in the state of the pulmonary artery of the diseased lung have been noted in five cases. In one (XXIV.) its branches were said to be simply dilated, whilst in another case (XIII.), observed by Dr. Wilks, he thus describes its condition :—" The pulmonary artery was very much diseased. It was, in the first place, considerably dilated, the branches throughout the tissue being much larger than natural. The coats of the vessel were also very much thickened, and the whole under surface was covered with atheromatous deposit. The vessel, in fact, very much resembled a diseased aorta. Some of the smaller branches were entirely obstructed by ante-mortem coagula, as were also some of the pulmonary veins. In the main pulmonary vessel there (as a layer of fibrine closely adherent to the wall, and with difficulty separable." Of the remaining three cases, in one (X.) the pulmonary artery was *contracted* to about the size of the coronary artery, whilst within it was a mass of fibrine which occupied the entire course of the artery even to its smaller branches, and at the same time was continuous with an adherent fibrinous mass in the right ventricle, in another (XII.) the pulmonary artery " seemed to be quite contracted ;" and in the last (XXII.) the vessel is not stated to have undergone contraction, but to have been completely filled with firm laminated colourless fibrine which adhered to its walls.

In four cases the bronchial glands were enlarged, and had become more or less indurated from a fibroid infiltration of their texture.

In only four[2] out of the thirty cases I have tabulated was there any fibroid induration of the opposite lung, and, except in one of these

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[1] After describing the lining membrane as smooth or granular, generally of a dark-red colour, and as almost invariably thickened, Barth adds : " Mais ce qui les distingue particulièrement, ce sont des espèces de stries irrégulièrement circulaires qui apparaissent plus ou moins distinctement sous la membrane interne, laquelle se continue manifestement avec la membrane muqueuse des conduits aëriens."—Loc. cit. p. 511.

[2] Nos. II., XIII., XXV., XXIX.

cases (II.), it was very small in amount, forming only two or three patches. In almost every case the lung of the opposite side was enlarged, and sometimes to a very considerable extent; it being mostly soft and crepitant throughout, and occasionally emphysematous. In many cases it extended as far as and beyond the opposite border of the sternum, and in one case (XXIX.) where its development was most extreme, it was just double its natural size, and seemed to fill almost the whole thoracic cavity.[1] In three cases only, as previously stated, was any "tubercle" found in the non-cirrhosed lung, and in these it was small in quantity. In one (IX.) there was a "tubercular" cavity in the apex, about the size of a walnut, filled with a coagulum of blood, death having been produced by a severe hæmoptysis; in another (XII.) "a few tubercles existed," and in the third (V.) a few "grey granulations" were said to have been scattered throughout the lung. As a rule, the only morbid characters belonging to the enlarged lung were those characteristic of the acute bronchitis, complicated with more or less of recent pneumonia—conditions which had been the immediate cause of the patient's death.

In nearly all the cases where the contraction of the lung had been great there was a proportionate traction of the heart out of its normal position. Where the right lung was involved, the heart was frequently found behind the right mammary region, and its displacement seemed generally to be greater where this lung was affected than when the left organ was the seat of the disease. Cirrhosis of the left lung tends to raise the heart, and in one remarkable case (XII.) reported by Dr. Law, it was found immediately under the left clavicle. In no less than ten out of the thirty cases there was more or less hypertrophy with dilatation of the right cavities of the heart, and in seven of these more or less dropsy also existed; whilst in two cases only (VIII., XXX.) was the heart reported to be rather small. In one case (X.) it was fatty, and in two of those in which the right ventricle was hypertrophied (XXIV., XXV.) the left was said to be small and weak.

PATHOLOGY.—Various views have been entertained as to the pathology of this affection, to which we must allude before entering more fully into the relative importance of those having the most decided claim to recognition.

(*a.*) Laennec first attracted attention to the disease, and considered it to be one of the modes in which dilatation of the bronchi occurred. He believed that chronic catarrh, giving rise to an increased secretion from the bronchial tubes, caused an accumulation of mucus within them, which led to their dilatation. The dilated bronchi, by pressure upon the surrounding lung-tissue, then led to its collapse and condensation.

(*b.*) Dr. C. J. B. Williams[1] held that it was the sequence of a pleuro-

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[1] The Pathology and Diagnosis of Diseases of the Chest, 1840, p. 99: his first allusion to the affection being in "Lectures" published in the Med. Gaz. for 1838.

pneumonia. His words were : " In pleuro-pneumonia the lung is inflamed, and at the same time compressed by an effusion in the sac of the pleura. If it remains long in this state, the smaller air-tubes and cells become obliterated by the adhesion of their sides, so that when the liquid is removed from the pleura they will not expand again with the enlargement of the chest ; but the large and middle-sized bronchi are not obliterated ; they bear the whole force of the inspired air, and become consequently dilated by it. This kind of dilatation is usually conjoined with contraction of the affected side. These cases, although not very uncommon, were first noted by the writer." And in a note to a subsequent work he said : " Dr. Corrigan has since described cases which appear to be similar, although he has given the disease the name *Cirrhosis* of the Lung."

(*c.*) Sir D. Corrigan maintained that the obliteration of the air-cells and condensation of tissue were primary, and were owing to the growth throughout the organ of a fibre-tissue similar to that existing in cirrhosis of the liver. The dilatation of the bronchi was a secondary effect, due partly to the greater stress of the inspiratory force, and partly to the traction, in different directions, exercised upon the tubes by the contracting fibre-tissue.

(*d.*) By M. Grisolle, M. Charcot, and others, what may be considered the early stages of this disease have been supposed to be the results of a " chronic pneumonia," or inflammation of the lung-tissue.

(*e.*) Dr. Hughes Bennett seems to ignore Cirrhosis of the Lung as a substantive disease, and to maintain that all cases of so-called Cirrhosis are, in reality, instances of tubercular disease advancing towards a cure.

Laennec's theory seems to be quite inadequate to account for the production of such a disease as Cirrhosis. And with regard to the second theory—that of Dr. C. J. B. Williams—it cannot be considered to apply to the class of cases to which Sir D. Corrigan gave the name Cirrhosis of the Lung. The mode of origin of these latter, as subsequent examination has fully shown, is entirely different ; the contraction of the lung being produced quite independently of the compressing effects of an effusion into the pleura.

Although Dr. Hughes Bennett is quite right in the view that there are certain cases in which a cirrhotic process is associated with " tubercle " (chronic lobular pneumonia) in the same lung, and in the opinion that this combination may, very rarely, terminate in a result differing but slightly from what may be produced by the pure cirrhosing process, still, what has been already said fully shows that in many cases Cirrhosis of the Lung is an independent affection, having no relation whatever to the presence of " tubercle " in the organ.

The relations of Cirrhosis to the common forms of Phthisis will be immediately considered ; and also the anatomical affinities between the tissue-changes in this disease and those which characterise Tubercle. The real relationship existing between dilated bronchi and Cirrhosis, will also be carefully considered. These questions will be

discussed under the following heads:—1. The Relations existing between the Cirrhosing Process and Chronic Lobular Pneumonia. 2. The Anatomical Affinities between the Early Stages of Fibroid Indurations and Tubercle. 3. The Mode of Production of Dilatation of the Bronchi, and their Relations to surrounding Induration of Tissue.

1. *The Relations existing between the Cirrhosing Process and Chronic Lobular Pneumonia.*—The evidence I have brought forward in the last section seems to show very conclusively that the cirrhosing process as it invades the lungs has no *necessary* connection with the development of "tubercle" in the same organ, whilst other considerations seem to show just as conclusively that its occurrence is not dependent upon the presence of a "tubercular diathesis." In four only, out of the thirty cases of Cirrhosis, did any morbid product which the observer was able to call "tubercle" exist in one or other of the lungs, in company with this fibroid conversion; and even in these cases the amount of the product (which most pathologists now consider as the anatomical mark of "Chronic Lobular Pneumonias"[1]) was so slight as not to interfere with our belief that its presence was an accidental rather than a necessary element of the disease. There is nothing antagonistic between these two pathological changes,—far from it. There cannot be a doubt, however, that each may, and does, exist by itself[2] as an independent affection, although they are so frequently combined in ordinary cases of phthisis—which differ from one another principally in respect to the relative proportion, and different modes of distribution, of these two tissue-changes. In proportion to the number of times in which the two processes are met with in combination, however, it may fairly enough be considered somewhat exceptional for either of them to exist, to a fatal extent, alone.

The facts at present known seem fully to establish the independent nature of the fibroid change met with in Cirrhosis. The word Phthisis is now generally admitted to be merely a generic term, under which are included different morbid conditions of the lung, which may either exist alone or in various degrees of combination. Thus, amongst other forms, there may be an almost pure pneumonic phthisis, due to the infarction of the air-cells and minute bronchi with epithelial products, the whole mass of which rapidly degenerates, and may break down into ulcerous cavities;[3] or a pure tubercular

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[1] In only one of the cases is there any mention made of the existence of "grey granulations," and in this case their nature is more than doubtful, since no similar granulations were found in any other organ.

[2] Occasionally, in some cases of "galloping phthisis," both lungs may at the autopsy be found thickly studded from base to apex with soft patches of "lobular pneumonia." These patches, of the size of a mustard-seed and upwards, are whitish or yellowish, breaking down here and there into minute cavities, whilst there may be a singular absence of all indurating tissue.

[3] In these cases the amount of new fibre-tissue is reduced to a minimum. Some slight amount, however, always exists, even in situations where there is no perceptible induration. The very early stages of the fibre overgrowth, when it is principally in a nuclear condition, do not produce indurations of the organs in which it occurs.

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Where the process of fibroid substitution is advancing in a lung, there appears to be not only an increased growth of the connective tissue and lymphatic elements, but also a rapid formation of epithelial products, as evinced by the number of cast-off and fattily degenerated cells of this kind which are seen within the air-vesicles. These are always to be seen in places where the fibro-nuclear growth has not completely invaded the tissue, though after a time they appear to be stifled, and stamped out as it were, by the superior energy in growth of the advancing fibre-tissue. This, in fact, appears to be the rule in pathological conditions of the lung, that a morbid change is rarely or never absolutely restricted to one tissue. The change originates and is predominant in one, whilst it extends to and may be only more or less slightly developed in the other. The nutrition of the organ, or of parts of it, may be generally deranged, but the stress of the disorder falls in one case principally upon the vascular province of the pulmonary artery, and in another upon that of the bronchial arteries: thus a bronchitis or a catarrhal pneumonia may be associated with a certain amount of fibroid induration, and an advancing fibroid change is often mixed up with an increased growth and shedding of epithelial elements from the mucous membrane.

Such being the anatomical nature and mutual relations of these various tissue changes, in what light should we regard the one with which we are now concerned—that which has been spoken of successively under the names of grey tubercular infiltration, chronic pneumonia, and fibroid induration or degeneration? That it is tubercular, or in any way an essential appanage of the tubercular diathesis, may, I think, at once be dismissed from consideration, as there is no evidence to support this view.[1] Is it then an inflammatory change, or one par-

In acute cirrhosis in the human subject there is often a slight tendency to extension of the process to other organs, and this tendency becomes more marked and constant in the rodent animals, though the spread to other organs is distinctly successive, and seems to take place by actual local contaminations. The frequency of cheesy degenerations in the infiltrating patches of "artificial tubercle" is probably referrible in the main to their rapid growth, and the ... bility of tissue elements which this usually entails.

[1] When the above passage ... n, I could speak thus confidently; now, however, since the experimental resear... ..., Wilson Fox, Dr. Sanderson, and others, upon the "Artificial Production of ... pathological doctrines show signs of undergoing ... modification. In the art... referred to, in the Edin. Med. Journ. 1869, Dr. ...son's view is most clear... It seems ... In this form: "Tubercles are ...d bodies enlarged: ... ... pr... by continuous growth, but ...stribution or dispersion ... point." For the develop-... "consumption" in ... ...1. A constitutional ten-A local irritation; and ... Referring to the latter, ... says: "The word ... r a chronic induration, "...ted corpusculation, ... give rise to similar ...ve." Dr. Sanderson ...to the mode of exten-...illed infiltrated ... in ordinary cases of ...could, of course, ...iltrating indurations ...) generally, ...haracterised by an ...iation" in th... ...lency, with some ...r that all ... ...ny increase by a ...be logical ... ...elief that each ...miliar... ...y writers would ...Sanderson ...ar hyperplas

since pathologists have begun to recognise the fact that, if the word
"tubercle" is to be preserved[1] at all, the grey granulation of Acute
Tuberculosis must be considered as its type. This alone of all the
morbid products which have been so named has a definite constitution
in whatever organ of the body it may be encountered; whilst the so-
called "crude tubercle," and cheesy products generally, may have had
the most diverse origin in different cases, and are always nothing but
the dead and impacted remains of various secretions and tissues. An
examination of very thin transverse sections of grey granulations in
the lungs, brain, liver, kidneys, and other organs, suffices to convince
one that its structure—closely allied to that of lymphatic tissue—
is always that of a small fibro-nuclear tumour infiltrating and sup-
planting the normal tissues of the part in which it is found. But,
further, it seems to me that the structure of tubercle is almost indis-
tinguishable from that of the tissue existing in the first stage of that
state which I have just been describing—hitherto known by the
various names of "grey tubercular infiltration," "chronic pneumonia,"
or "fibroid degeneration." There are in each case the same round
and oval nuclei or embryo-cells, imbedded in a fine and somewhat
scanty fibrous stroma. This resemblance only exists, however, be-
tween one temporary stage of the process of fibroid substitution, and
the grey granulation.[2] Tubercle seems to be the mark or index of a
general constitutional disease, and how long the grey granulation may
remain as such, or what may be the degree of frequency with which it
undergoes changes, are questions to which we are unable to give very
satisfactory answers. Although fibroid induration may, on the other
hand, owe its origin partly to a constitutional cause, it seems much
more dependent upon special local conditions operating in the organ
or part in which it is set up; then again, it exists not only in minute
patches, but spreads over considerable areas, and advances through
stages of development which are well known and pretty constant.[3]

[1] For my own part, I think that pathological science would gain much if this word,
and all the erroneous associations, as to specificity, which its use seems inevitably to
entail, could be entirely forgotten, save as errors of the past. Old things might receive
new names, and thus, at last, old theories might possibly be shelved.

[2] It is interesting to find that, nearly forty years ago, Andral seems to have anticipated,
in a measure, the results of recent microscopical research, since he fully recognised that
the grey granulation was quite distinct from other kinds of what was then called
"tubercle," and was closely allied rather to the forms of pulmonary induration which
we now know to be of fibroid origin. His words were (Précis d'Anat. Patholog. 1829,
t. ii. p. 518) : "Les granulations pulmonaires de Bayle ne sont ni un tissu accidentel
sui generis, comme il le pensait, ni le premier dégré du tubercle comme l'ont admis
MM. Laennec et Louis, mais qu'elles consistent dans l'induration de quelques vésicules
. . . . Or, ce qui arrive à un lobe dans sa totalité peut aussi arriver à quelques vési-
cules ; la lésion est seulement moins étendue ; mais du reste, sa nature est la même."

[3] It seems the so-called "artificial tubercle" in the rodent animals whose anatomical
characters have now been fully revealed by the admirable researches of Dr. Sanderson, Dr.
Wilson Fox, and others, is less allied to tubercle (as occurring in acute tuberculosis) than
to some more local manifestation, such as that which characterises "tubercular perito-
nitis." All these morbid products are, however, as I think, more akin to those of acute
cirrhosis. In acute tuberculosis, as it occurs in the human subject, the grey granula-
tions appear to develop almost if not quite simultaneously in meninges, lungs, liver, &c.

Where the process of fibroid substitution is advancing in a lung, there appears to be not only an increased growth of the connective tissue and lymphatic elements, but also a rapid formation of epithelial products, as evinced by the number of cast-off and fattily degenerated cells of this kind which are seen within the air-vesicles. These are always to be seen in places where the fibro-nuclear growth has not completely invaded the tissue, though after a time they appear to be stifled, and stamped out as it were, by the superior energy in growth of the advancing fibre-tissue. This, in fact, appears to be the rule in pathological conditions of the lung, that a morbid change is rarely or never absolutely restricted to one tissue. The change originates and is predominant in one, whilst it extends to and may be only more or less slightly developed in the other. The nutrition of the organ, or of parts of it, may be generally deranged, but the stress of the disorder falls in one case principally upon the vascular province of the pulmonary artery, and in another upon that of the bronchial arteries : thus a bronchitis or a catarrhal pneumonia may be associated with a certain amount of fibroid induration, and an advancing fibroid change is often mixed up with an increased growth and shedding of epithelial elements from the mucous membrane.

Such being the anatomical nature and mutual relations of these various tissue changes, in what light should we regard the one with which we are now concerned—that which has been spoken of successively under the names of grey tubercular infiltration, chronic pneumonia, and fibroid induration or degeneration? That it is tubercular, or in any way an essential appanage of the tubercular diathesis, may, I think, at once be dismissed from consideration, as there is no evidence to support this view.[1] Is it then an inflammatory change, or one par-

In acute cirrhosis in the human subject there is often a slight tendency to extension of the process to other organs, and this tendency becomes more marked and constant in the rodent animals, though the spread to other organs is distinctly successive, and seems to take place by actual local contaminations. The frequency of cheesy degenerations in the infiltrating patches of "artificial tubercle" is probably referrible in the main to their rapid growth, and the instability of tissue elements which this usually entails.

[1] When the above passage was written, I could speak thus confidently ; now, however, since the experimental researches of Dr. Wilson Fox, Dr. Sanderson, and others, upon the "Artificial Production of Tubercle," pathological doctrines show signs of undergoing some modification. In the article before referred to, in the Edin. Med. Journ. 1869, Dr. Sanderson's view is most clearly stated. It comes out in this form : "Tubercles are adenoid bodies enlarged : . . . the disease progresses, not by continuous growth, but by the distribution or dispersion of infective material from one point." For the development of "consumption" in man, three things are necessary :—1. A constitutional tendency ; 2. A local irritation ; and 3. A process of *infection*. Referring to the latter, Dr. Sanderson says : "The word designates the fact that wherever a chronic induration, *due to overcrowded corpusculation*, exists in any organ, it is apt to give rise to similar processes elsewhere." Dr. Sanderson would apply these views even to the mode of extension of "the so-called infiltrated forms of induration" met with in ordinary cases of phthisis ; and he would, of course, be compelled to apply it to infiltrating indurations of cirrhosis processes) generally, because they are almost always characterised by an "overcrowded corpusculation" in the part. Thus the present tendency, with some pathologists, is to consider that *all* infiltrating fibroid indurations may increase by a process of infection, and the logical outcome of their doctrines is the belief that such indurations are tubercular in nature. The chronic inflammations of many writers would thus be transmuted into "tubercular" affections, and the simple nuclear hyperplas

taking rather of the nature of a degeneration? To Dr. Handfield Jones the merit is due of having first fully pointed out[1] the essential similarity of these indurating processes in various organs of the body (all of which had been previously spoken of as effects of "chronic inflammation"), of having shown that in all alike the essential nature of the change is an hyperplasia or overgrowth of the connective tissue of the part, and for ably insisting that the process by which this was brought about was one totally distinct from what is ordinarily understood by the word inflammation. He held that they were effected, in fact, by a process substantially different—by one which was slow and chronic from the first, and which partook rather of the nature of the process by which an infiltrating new growth spreads.[2] It seems to me, also, that the word inflammation is quite inapplicable to the changes by which these effects are brought about. In inflammation we almost invariably find an accelerated formative process resulting in the production of elements of an unstable composition, such as quickly degenerate and decay—a process of necrobiosis or destruction in fact goes on simultaneously with one of formative increase—whilst in the process which results in the production of fibroid indurations, there is principally an increased formative stimulus by which an overgrowth of connective tissue or lymphatic elements takes place. The necrobiotic process, however, is almost entirely wanting, since the new-formed elements persist as a developing fibroid growth. Thus, whilst the change differs materially from inflammation, so also does it differ from a degeneration. The proper tissues of the part are not merely degenerated and structurally spoiled, they are actually killed, and disappear before a new fibro-nuclear tissue which supplants them. So that we have the increased formative energy of an inflammatory process without its unstable products; and we have the functional degradation characteristic of a degeneration—though this results not from mere spoiling of texture, but rather from the complete substitution of a tissue of a lower grade in the place of that which is proper to the part. Surely in this fibroid hyperplasia, or *fibroid substitution*, as I think we should term it, we have a process strictly intermediate in kind between inflammation on the one hand, and degeneration on the other—it is a sort of neutral ground from which the other two processes may be considered as divergences in opposite directions.[3]

3. *The Mode of Production of Dilatation of the Bronchi, and their Relations to surrounding Induration of Tissue.*—The opinions expressed as to the mechanism of dilatation of the bronchi have been most various since the subject was first introduced by Laennec. His theory was, that bronchial dilatation was one of the effects of chronic

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which characterise them in their early stages would be even more likely to be considered as a new "specific product," if it is to receive the name of "adenoid" tissue.

[1] Brit. and For. Rev. 1854. This, as we have seen, is also the opinion which was subsequently expressed by Dr. Wilks. [2] Loc. cit. p. 345.

[3] The phrase "fibroid substitution" will not be applicable to all instances of the kind of change alluded to, since, where it occurs in some of the fibrous membranes, such as the arachnoid, there is no substitution, but only an increase or hyperplasia of the part.

bronchial catarrh—that it was brought about by the accumulation and stagnation of mucus in the inflamed tubes, and that the dilatations, by the pressure they exercised, led to the collapse and consolidation of the surrounding lung tissue. Andral's views[1] were also somewhat unsatisfactory. He recognised three forms of dilatation: one species, with thin walls, he believed was produced after the manner stated by Laennec, whilst two others he attributes to hypertrophy of the bronchial walls, though he does not explain how the modification in texture is to bring about the alteration in calibre of the tubes. Dr. Stokes[2] believed bronchitis to be in all cases the primary cause of the dilatations, inasmuch as this leads to loss of elasticity in the longitudinal contractile fibres of the bronchi, and also to paralysis of the circular muscular fibres. He thought also that the epithelial ciliary action ceased, and thus permitted the accumulation of mucus, which (in conjunction with the other causes mentioned) tended to bring about a dilatation of the tubes, under the straining influence of forced inspirations, during repeated attacks of coughing. Dr. C. J. B. Williams[3] also laid great stress upon the influence of inflammation in bringing about alterations in the texture of the tubes, by which their elasticity and power of resistance was impaired—so that they more easily yielded to pressure during the act of coughing. This was his theory as to the mode of production of the ordinary forms of bronchial dilatation—those which exist without great induration of the surrounding lung texture. Where extreme induration was also present, however, he gave the explanation which has been quoted at the commencement of this section. (See p. 818).

Very shortly afterwards Sir D. Corrigan[4] published his explanation of the production of bronchial dilatation, as met with in the class of cases to which he gave the name Cirrhosis of the Lung. This must be given in his own words. He says: "The dilatation of the bronchial tubes is partly owing to the contractile process going on in the tissue of the lung—partly to the expansive action of the parieties of the chest in the act of inspiration. . . . . . . If there were but one bronchial tube with contracting fibro-cellular tissue placed around it, then the contracting tissue would, as in the instance of stricture of the æsophagus or rectum, cause narrowing of the tube; but when there is, as in the lung, a number of bronchial tubes, and the contracting tissue not placed around the tubes, but occupying the intervals between the tubes, then the slow contraction of this tissue will tend to draw the parietes of one tube towards the parietes of another, and necessarily will dilate them." He also says: "In proportion as the contraction of the fibro-cellular tissue obliterates the small air-vesicles, and as these contracting fibres, like so many strings, extending from the root in all directions, tend to contract or draw in the tissue of the

[1] Précis d'Anat. Patholog. tome ii. p. 496.
[2] Diagnosis and Treatment of Diseases of Chest. Dublin, 1837.
[3] Pathol. and Diagn. of Diseases of the Chest, 1840, p. 96.   [4] Loc. cit. p. 270.

lung, obliterating its small air-tubes and its blood-vessels, the larger bronchial tubes dilate to supply the place thus left, until, when the disease has reached its last stage, the tissue of the lung, diminished to a very small size, presents no longer any permeable air-vesicles, but a dense fibro-cellular or fibro-cartilaginous tissue with its fibres radiating in every direction, through the second and third sized bronchial tubes, dilated into cells, or ending in *culs de sac*, of every variety of size."

Rokitansky[1] adopted Dr. Stokes's view as to the mode of production of the uncomplicated form of bronchial dilatation: he believes it to be a result of obstructive bronchitis in the ramifications of the bronchi beyond those which become dilated. "It is produced," he says, "by the hindrance which is presented to the free ingress of the inspired air, and is proportional to the difficulty of breathing, and the prolonged length of each individual inspiration, and is especially developed in and about the perfectly impermeable bronchial tubes. The parenchyma surrounding this portion of the bronchial system collapses, and this produces a space which becomes filled by the dilating bronchus. The dilatation thus lies entirely, or for the most part, in a collapsed, and apparently compressed, portion of the parenchyma; hence the latter appears to be the primary anomaly, and the bronchial dilatation merely a *resulting* and consecutive morbid change." The opinions expressed by Dr. Gairdner[2] were very different, and are as follows:—"The conclusions to which I have been led by this survey is, that almost all the so-called bronchial dilatations, and all those presenting the abrupt sacculated character here referred to, are in fact the result of ulcerative excavations of the lung communicating with the bronchi." He then adds: "The usual origin of bronchial dilatations is in cavities formed in atrophied lungs, in consequence of bronchitis or tubercle, and afterwards expanded beyond their original dimensions by the inspiratory force." Dr. Peacock[3] thinks Sir D. Corrigan's views unsatisfactory, but he says, in reference to the views of Dr. Williams and Dr. Gairdner: "I believe both to be correct in some cases, and that by one or other of the modes mentioned by these writers all the various forms of so-called dilatation of the bronchial tubes which are observed may be explained." M. Barth[4] believes to a certain extent in the views advanced by Stokes, and also partly in those of Corrigan—to the effect that condensation of tissues usually precedes the bronchiectasis. He also attributes an influence to firm pleuritic adhesions when combined with a shrinking of lung-tissue, and to the pressure exercised by retained and heated air which has been forcibly drawn, through accumulated mucus, into certain bronchi.

Lebert[5] agrees, in the main, with Stokes, though he thinks the

[1] Pathol. Anat. (Syd. Soc. Trans.).
[2] Monthly Journal of Medicine, vol. xiii., 1851, pp. 248, 249.
[3] Ibid, April 1855, p. 285.   [4] Loc. cit. p. 517.
[5] Anat. Patholog. tome i. p. 620.

weakness of the bronchial walls is ultimately dependent rather upon a disturbance in their innervation than upon an inflammatory state. Quite recently, Dr. Grainger Stewart[1] has objected to the theory of Stokes, urging that if bronchiectasis depended simply on bronchitis, it would necessarily be much more frequent than it is. He thinks that Lebert's doctrine is the only one which is not opposed to known facts, and draws the following conclusions from his own observations:—" 1. That the essential element of bronchiectasis is atrophy of the bronchial wall, that the cause of such atrophy is not yet ascertained, but may perhaps be connected with constitutional peculiarities. 2. That the walls being so thinned and weakened, readily yield to the pressure of air, it may be in deep and sudden inspirations or during violent muscular exertions, certainly in the sudden expiratory effort made while the glottis is closed in the act of coughing. 3. The enfeebled and dilated condition of the bronchi favours the accumulation of the mucus secreted by the bronchial membrane. 4. That the mucus accumulating and undergoing decomposition in the dilatations, irritates the mucous membrane, leads to inflammation, and the formation of villous processes from it, to the formation of increased connective tissue in the walls, to irritation of the cartilages, and frequently to consolidation of the surrounding lung-tissue and pleuritic adhesions, sometimes also to abscess or to limited gangrene." With regard to the primary atrophic change which takes place in the walls of the bronchi, Dr. Stewart says that this is obvious even in the slighter dilatations, in which the mucous membrane is as yet unaffected, and that the atrophy shows itself in the muscular and elastic fibres, which appear granular and indistinct.

Such are the various opinions that have been expressed concerning the mechanism of bronchiectasis, and the relations of this pathological condition to surrounding induration of lung-tissue; and one cannot help being struck with the very opposite views which certain of the writers take as to the interdependence of these two states. This very diversity of opinion, however, seems to indicate that condensation or induration of lung-tissue cannot in all cases be considered as a necessary prelude of bronchiectasis. Those who have formed this opinion must have arrived at their conclusion from an examination of a limited class of cases, since it is a well-known and admitted fact to those who have studied the subject more widely, that in certain cases dilatation of the bronchi exists with scarcely any appreciable alteration of the surrounding lung-tissue. But whilst in some cases it seems certain that adjacent induration either does not exist, or is present to such a limited extent as to be altogether unimportant in an etiological point of view (even if, in these cases, it has not been mechanically produced by the very dilatation with which it co-exists), it seems also just as evident that, in a certain class of cases, the bronchial dilatation is to be looked upon as a secondary consequence of induration and contraction of the lung-tissue. What the precise

[1] Edinburgh Monthly Journal, 1866.

means are by which the dilatation is brought about in these cases, we shall consider presently; but that the existence of a disease of the lung-tissue, which entails contraction, is favourable to the occurrence of bronchial dilatation, may be seen, I think, from the facts before mentioned,—to the effect that nineteen or nearly two-thirds of the thirty cases of Cirrhosis I have analysed, occurred in individuals between the ages of fifteen and forty years, and that, out of these nineteen cases, eleven presented well-marked dilatation of the bronchi; whilst in the forty-three cases of dilatation of the bronchi collected by Barth, only seven—or less than one-sixth of the total number— were met with between these ages, though more than one-half (26 : 43) were in individuals over sixty years of age. The occurrence of Cirrhosis of the Lung, therefore, seems to be favourable to the production of bronchiectasis at such ages when dilatation of the bronchi alone, or as a primary phenomenon, is not prone to occur.

With reference to the occurrence of bronchiectasis in lungs which are not contracted, and have no consolidation of tissue in them, it seems to me that if a primary atrophy of the bronchial walls exists like that which Dr. Grainger Stewart has observed, the order or succession of the phenomena would probably be such as he describes. This mode of origin, also, seems to be the only one capable of accounting for such cases of bronchiectasis as have been met with unexpectedly, in individuals who have not had any long-continued cough or bronchitis: it may, moreover, obtain in the first instance, and be the determining cause of the dilatation in a certain number of those persons who have previously suffered from bronchitis. By reference to such a mode of origin only, does it seem possible to explain some of the anatomical characters of dilated bronchi, such as the occurrence of bridge-like portions of prominent and unatrophied tissue, and the occasional communication between the dilated portions of contiguous tubes. But it seems equally plain that it is not necessary for us, in all cases, to assume the existence of such an atrophy, when we recollect in what a large proportion of cases the individuals in whom bronchiectasis has been met with have suffered from chronic bronchitis and long-continued cough. To explain the occurrence of dilated bronchi in many of these cases, we have only to refer to the views of Dr. Williams and Dr. Stokes, before alluded to; and I would also add, that one important kind of alteration in the walls of the bronchi, induced by chronic inflammation, is the production of a certain amount of fibroid substitution. Then, as in most of the cases of dilatation of portions of the vascular system, more or less of the muscular and elastic tissue of the tubes is replaced by ordinary distensible, though comparatively unelastic, fibrous tissue.[1] A tube thus altered, having once yielded under a powerful inspiratory effort,—

[1] Dr. Stewart says that many of the dilated bronchial tubes present an appearance simulating hypertrophy of their walls, but which is really dependent upon changes in the mucous membrane, by which it becomes granular or vilous, and upon the presence of ill-formed connective tissue among the denser elements of the bronchial walls. He adds, "The irritation which causes the inflammatory thickening of the mucous coat may well also account for the spurious hypertrophy of the other."

or more especially under the powerful expiratory effort, with closed glottis, preceding the act of coughing,—does not regain its normal calibre, and each increment of dilatation successively brought about remains as a persistent abnormality. In those instances of what may be called acute dilatation of the bronchi, met with after attacks of hooping-cough, the inflammatory changes in the walls of the tubes, combined with the powerful inspiratory and expiratory efforts, seem to be the conditions which are most instrumental in bringing about this effect. Then again, the modes of origin suggested by Dr. Gairdner must not be forgotten. There seems every reason to believe that many of the abruptly saculated cavities which have been described as bronchial dilatations, have really had an ulcerative origin, though their walls may have become perfectly smooth. Cavities thus formed may subsequently be increased in volume by the same means as those which usually suffice to augment the size of the more simple bronchial saculi.

Although in a certain number of cases little or no alteration of the lung-tissue around the dilatations exists, in many others more or less condensation is met with. This is oftentimes merely a collapse of the adjacent textures, brought about by the pressure of the dilating bronchus; whilst, in other instances, there is an actual induration of tissue, which must be regarded as a consequence of the primarily existing bronchial dilatation. Dr. Grainger Stewart has suggested what may be considered to be a real and feasible explanation of this secondary induration in his fourth conclusion, where he says that influences which suffice to irritate the bronchial wall must, if continuously or intensely applied, affect the structures lying beyond them. In one case, around the dilated bronchi he found the lung-tissue indurated and pneumonic; and in another case, around cavities which were livid with reddened and inflamed mucous membrane, the lung-substance was consolidated. On microscopical examination of this consolidated lung-tissue, "little trace of air-cells could be made out, and it was mostly composed of fibrous tissue." In other rare cases, the irritation manifests itself in the formation of an abscess, in the centre of which the dilated bronchus is seen; or even—as first pointed out by M. Briquet [1]—in the establishment of a limited gangrenous inflammation, involving the walls of the dilated bronchus and the surrounding lung-tissue.

In other instances, where the bronchiectasis is primary, instead of the intervening lung-tissue remaining unaltered, being simply compressed, or undergoing either of the secondary changes just mentioned, it gradually disappears—seemingly as a result of atrophy and slow absorption—so that, in extreme cases, absolutely no intervening tissue may be left between the dilated tubes of the greater part of one lobe of a lung.[2]

[1] Archives Générales de Médecine. 1841.
[2] There is a good example of this in Guy's Hosp. Museum, 1718⁶¹. See also Path. Trans. vol. xii. p. 78.

We must now come to a consideration of the mode in which dilatation of the bronchi is brought about in Cirrhosis—that is to say, in those cases where induration and contraction of the lung-tissue is the primary occurrence, and where dilatation of the bronchi is an altogether secondary phenomenon, which may occur or may not, according to the presence or absence of other occasional accompaniments of the disease. An analysis of the thirty cases I have tabulated seems to show that dilatation of the bronchi in this disease is of a compensative character, owing to the fact of its being generally most marked in those contracted lungs where the space which would have been left by contraction is not otherwise filled up—either by inshrinking of the thoracic parietes, by elevation of the corresponding half of the diaphragm with proportionate displacement of abdominal organs, or by hypertrophy of the opposite lung and its extension into the diseased side of the thorax. If the space which would have been left by the shrinking lung is not otherwise filled up, then the increased pressure of the inspired air, acting upon bronchi in whose walls more or less fibroid substitution has most likely occurred, tends to dilate some of those which are most favourably situated for undergoing this expansion. It is obvious that something must go towards filling up the space left by the shrinking lung; and if the thoracic parietes are so firm as not to yield easily, or if displacement of the viscera does not take place, then the bronchi must yield and dilate in some of their weakest parts under the continually increasing pressure of the inspired air. It is, however, in great part a mechanical question. In a case where the proper texture of the tubes has become weakened by inflammation or fibroid changes, and where other conditions are favourable, a dilatation may be brought about; whilst in another case, where the lung is equally affected, dilatation of the bronchi may not occur, because in this particular instance, it may be easier for displacement of viscera or inshrinking of the thoracic parietes to occur in its stead. Of course this dilatation need not necessarily be situated—and in fact would be less prone to occur—in parts of the lung which had already undergone an extreme amount of induration. So long as the dilatation existed in some part of the organ, the particular region in which it occurred would be altogether immaterial. The weakest part, other things being equal, would most readily undergo dilatation. How far the contractile influence of the fibre-tissue itself may, as suggested by Sir D. Corrigan, directly tend to bring about the dilatation of the bronchi, or be a real cause of their enlargement after a certain amount of dilatation has once been established, seems doubtful. I certainly do not think, however, that this is one of the principal causes of the production of bronchiectasis. If it were really the method by which dilatations of the bronchi had been produced, it might reasonably be expected that they should be most marked precisely in those parts of the lung which had undergone the most notable contraction and condensation. Such a distribution is, however, by no means invariable, and often the arrangement met with is quite the reverse.

Taking the view of the case I have proposed, it will be seen that in those instances where dilatation of the bronchi did not exist, or was only very slightly marked,[1] this was explicable from a consideration of other co-existing conditions observed *post mortem*. Thus, in two of the cases in which there was no bronchiectasis, the lung affection was comparatively acute and recent, and no shrinking of the organ had as yet taken place; in the next the amount of lung shrinking was probably not great, as no note was made of its existence; in another the lung was described as "small and solid" on the right side, but then the liver was very large, and the right side of the chest was also flattened; in another the disease was restricted to the lower lobe of one lung on the right side, but then this was universally adherent to the diaphragm, and the upper lobe of the same lung was notably emphysematous; whilst in the last, although the right lung was as small as a closed hand, there was flattening beneath the clavicle, and the right side of the thorax contained a large and displaced heart, in addition to nearly one quart of pleuritic fluid.[2] Of those cases in which the dilatation of bronchi was only slightly marked, in one the diseased lung was universally adherent, and its amount of shrinking was probably not extreme, since it was not specified, whilst there was a most remarkable dilatation of the pulmonary artery throughout the organ; in another, the disease being on the right side, there seemed to have been a falling in of the lower part of the thoracic parietes, whilst the heart was situated entirely in the right side of the thorax, and the enlarged left lung extended under the sternum and partly into the right side; in another, although the amount of contraction of the lung was extreme (the disease being of six years' duration, and having commenced when the boy was only fourteen years old), still the left side was described as being "contracted to an extraordinary degree," both vertically and horizontally; in the last the reason why there was only slight dilatation of the bronchial tubes is not quite so evident, though some of the points which might have explained it have not been distinctly alluded to. Concurrent evidence of this kind strongly tends to support the view now advanced concerning the method of production of the bronchiectasis which may occur in the course of Cirrhosis.

From these considerations as to the mode of production of bronchiectasis generally, and its relation to different states of the surrounding lung-tissue, we may venture to draw the following conclusions :—

1. That dilatation of the bronchi may be present, and take place quite independently of alterations in density of the surrounding lung-texture; although such dilatation may be favoured by a primary atrophy of the walls of the bronchial tubes, or by the effects of inflammation in weakening them and diminishing their natural elasticity, or by a combination of the two. The actual mode of production, even when these favouring conditions exist, being always the expanding

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[1] No dilatation of bronchi existed in Nos. II., III., VI., X., XV., XXII., and it was only slightly marked in Nos. XIII., XX., XXIII., XXV.

[2] This was the only one of the thirty cases, however, in which any pleural fluid was found, or in which there had been any reason to suspect its previous existence.

force of powerful inspirations, and more especially the tension occasioned by the expiratory effort, with closed glottis, which immediately precedes the expiratory part of the act of coughing.

2. That in these cases of *primary* bronchiectasis the intervening lung-tissue may be found almost natural, or compressed and airless, though it may subsequently become so far irritated as to be found in a condition of inflammation, of fibroid induration, of purulent softening, or even of gangrene.

3. That in certain other cases the bronchiectasis is compensative, and seems to be *secondary* to a certain amount of collapse of lung-tissue, though its actual production is still aided by the effects of cough and inflammation; or, as in so many of the instances of Cirrhosis of the Lung, the bronchiectasis is secondary to an actual shrinking with fibroid consolidation of the lung-texture—when dilatation of some of the bronchi results, as a physical necessity, if displacement of viscera or inshrinking of thoracic parietes cannot be so easily brought about.

ETIOLOGY.—Are we to look upon Cirrhosis of the Lung as a constitutional affection or as one of a strictly local nature ?   If constitutional, we should have to regard it as one of the local manifestations of a general diathetic condition upon which fibroid degeneration of organs and tissue seems in some cases to depend.[1]   The question of the existence or not of such a diathetic condition has been ably discussed by Dr. Handfield Jones,[2] who has shown that not unfrequently we meet with wide-spread degenerations of this kind existing in various organs of the body, which it seems only possible to explain by the assumption of the existence of some particular condition of the blood or diathetic state, favourable to the occurrence of such anomalies of nutrition in many parts of the same organism. Thus, coinciding with a cirrhosis of the liver, we may find a similar condition more or less developed in the kidney, together with opaque thickenings of the capsule of the spleen, fibroid thickenings of the cardiac valves, fibroid degeneration of the parts of the arterial system, opaque thickenings of the arachnoid, &c.   Do we in these cases meet with similar changes in the lungs, and is Cirrhosis of this organ to be looked upon as a sequence of the diathetic condition in question ?   To the first inquiry our answer must certainly be in the affirmative. Fibroid thickening and induration of parts of the lungs is frequently met with in association with similar changes in other parts of the body, as the tables of Dr. Sutton[3] fully prove.   An answer to the second question is, however, not quite so easy.

Although in a certain number of cases, and more especially in

[1] In the paper before alluded to, Dr. Andrew Clark has, since this was written, strongly urged that his cases of "Fibroid Phthisis" are local manifestations of a diathetic condition, characterised by the dissemination of waxy degenerations and fibroid indurations in different organs of the body.
[2] "Fibroid and Allied Degeneration," Brit. and For. Med.-Chir. Rev. 1854.
[3] Med.-Chir. Trans. vol. xlviii.

elderly persons, disseminated fibroid indurations are to be met with, still, in other cases, a notable amount of fibroid substitution may have taken place in one or other organ alone. This latter state of things, so far as I have seen, is more apt to occur in individuals who have not yet passed the meridian of life. But fully two-thirds, or perhaps more, of the cases of Cirrhosis of the Lung, are met with in individuals under forty years of age. Moreover, an examination of the post-mortem records of the thirty cases which I have tabulated, lends little or no support to the idea that the induration and shrinking of the lung has been only one manifestation of a general diathetic condition entailing similar changes in other organs. Again, it may be seen from a consideration of the pathology of that form of bronchitis which is set up by the continued inhalation of foreign particles, that a similar fibroid change may be initiated in the lungs, without the agency of any diathetic condition. By the powerful action of a local irritation only, such changes are set up and may be seen in association with the chronic bronchitis of miners and artisans. In these cases the determining cause acts upon both lungs, and the effects are seen in both. Not so, however, with the ordinary cases of Cirrhosis of the Lung: here the fibroid induration, when existing to the marked extent which constitutes Cirrhosis, is almost invariably unilateral (which of itself tends strongly to negative the idea of its being entirely of diathetic origin), and in only three or four out of the total number of cases does there seem to have been any well-marked co-existing fibroid substitution or hyperplasia in other organs. But we do find in more than two-thirds of the cases, old adhesions of a firm and almost cartilaginous consistence uniting the two pleural surfaces on the side affected.

Chronic bronchitis, in fact, when it occasions a dry pleurisy on one side with the gradual formation of adhesions, seems to be the most frequent determining cause of that local overgrowth of fibroid tissue which constitutes the essential feature of the disease. The new growth gradually encroaches upon and replaces the proper lung texture, till at last the whole nutrition of the organ seems to become leavened by this change, and many independent centres of transformation are established. In almost all cases, however, in which thickening of the pleura is produced, the new growth seems to spread inwards from this with greater rapidity than it does from other centres.

In a few cases chronic bronchitis alone seems to have been the determining cause, since no notable adhesions of the pleural surfaces have existed, and the invading new tissue has seemed to start, throughout the organ affected, as direct prolongations from the walls of greatly thickened bronchi.[1] Why in these latter cases the change should be limited to one lung is rather difficult to understand:[2] we can only

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[1] In very many cases this induration may never reach an extreme degree, and it may affect both lungs pretty equally. It is only in rare cases that it attains an extreme degree in one lung, and so produces the condition of the organ with which we are now concerned.

[2] A somewhat similar difficulty, however, presents itself in the case of simple dilated bronchi, owing to the frequency with which this condition, in association with chronic

flammation of the lung—the process which Grisol
other writers describe as " chronic pneumonia." This
rounded with doubt and difficulty. I have alread
name "chronic pneumonia" appears to me to be altog
and contradictory as applied to this affection. But,
from the question of names, there are other difficultie
owing to the rarity of the occurrence—many physici
pared to admit that such a pathological state is ever
sequence of an acute pneumonia. Several physicians
however,—such as Bayle, Sir John Forbes, Addison,
Charcot, Hughes Bennett, and others—believe in th
have recorded cases which tend most strongly to
opinion. Bayle's[1] case of "Chronic Peripneumony,
Phthisis," seems to be one of this kind. Grisolle says
very long experience, he has only seen four example
of acute into chronic pneumonia. He believes tha
is a consequence of neglect, though it may, perhap
more upon peculiarity and debility of constitution
is liable to occur in habitual drunkards, but Gris
such does not seem to be the case in France:
place any more credence in the opinion of Hesch
he found that this complication was rare at Vi
what more common at Cracow, attributed it to t
malaria. In one of Grisolle's patients, who died at
tenth week from the commencement of an acute pn
he considered to have passed over into the chronic
affected was found in the following condition :—It wa
hepatized; the lower lobe being hard, compact, re
the cut surface being smooth—though granulations
portions were torn. This differed from a state of act
by the greater hardness and grey colour of the par
the upper lobe was indurated, with the exception of a
ing rather more than one inch from the summit. The
which presented the most recent traces of inflammati

torn surface. This granular appearance may be met with, according to Grisolle, when the malady has only been of two or three months' duration, though after this it gradually disappears. Then, with regard to the case (II.) recorded by Dr. Sutton, although a previous history of inflammation of the lung was by no means distinctly made out, Dr. Sutton seems to have been quite convinced that the state of the organs was just such as has been described under the name " red induration," and it does appear quite certain that the change was one of an acute character. So that either the old interpretation must be the correct one (that this " red induration " is the immediate sequence of an acute pneumonia) or else we must accept Dr. Sutton's supposition, that it is possible for an acute fibroid change, of the kind he describes, to occur in a lung not previously diseased. But, in the face of other evidence, the first supposition seems the most probable one, and I look upon the case (III.) recorded by M. Charcot, as affording the strongest support to this view. Whilst believing, therefore, that this sequence may occur in certain cases, it must be clearly borne in mind that it is an occurrence of extreme rarity, supervening only under the influence of exceptional conditions, which as yet may be said to be almost entirely undiscovered.

In fine, then, exposure to cold and wet, leading to the advent of bronchitis, pleurisy, or pneumonia, in certain individuals, seems to be the principal determining cause of this disease. It is apparently much more prone to occur in males than in females, though this difference may, perhaps, be due more to the much greater frequency of exposure of individuals of the male sex, than to any inherent inequality in liability to the disease *quâ* sex. And, although met with occasionally in children and in old people, this disease seems much more prone to occur in individuals between the ages of fifteen and forty. But what has just been said with regard to the apparent determining influence of sex, and its subordination to relative amount of exposure to wet and cold, may also hold good with regard to age, since, *cæteris paribus*, individuals between the ages I have mentioned, are more likely to be exposed in this way, than persons who are either older or younger.

With regard to the supposed connexion between this disease and the rheumatic diathesis, or the predisposing influence of long-continued habits of intemperance, nothing positive can be said ; only the extreme rarity with which either of these circumstances has been mentioned would seem to show that neither of them can be considered as essential antecedents of the disease. No casual relationship, either, can be established between syphilis and Cirrhosis of the Lung. Neither does there seem to be any evidence to lead us to imagine that this malady is ever propagated by hereditary transmission : and of course if it be true, as I suppose, that the disease is a local one, set up for the most part in the individual by accidental conditions, this absence of any tendency to hereditary transmission is quite in accordance with what might have been expected.

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ANALYTICAL DETAILS OF THIRTY CASES OF CIRRHOSIS OF THE LUNG.

Sex, Age, and Observer.	General History.	Symptoms.	Inspection, Percussion, Auscultation, etc.	Autopsy.
I. M. æt. 7. Dr. Corrigan, Dublin Journ. of Med., 1858, p. 226.	Influenza three months before, followed by cough and expectoration, with loss of flesh, and occasional hæmoptysis.	Febrile symptoms for sixteen days, with severe cough, dyspnœa, and hurried respiration.	Right side perceptibly flattened. Bronchial respiration, and distinct bronchophony over flattened portion of chest.	*Right side*, slight pleuritis: lung solid, non-crepitant, greyish-red, tough, and traversed in all directions by thickened white bands of fibro-cellular tissue. Bronchi dilated towards pleura, terminating in spherical sacculi; lining membrane dark red. *Left lung*, healthy.
II. M. æt. 26, Dr. Barlow (recorded by Dr. Sutton), Med.-Chir. Trans., 1865, p. 299.	A well-developed, muscular man, of middle height. Always had good health, except for an occasional winter cold. Four months ago appetite began to fail and cough commenced. Pursued his work for one month, and then gave up, owing to increasing weakness. Afterwards became weaker and weaker, the cough continuing. Three weeks before admission spat phlegm streaked with blood.	*Oct.* 12.—Admitted into hospital. During the first ten days cough came easier, and he seemed to strength. Appetite variable. *Oct.* 21.—Immediately under clavicle scarcely any resp. heard, but distant crepitati... teriorly over right apex tubular breathing, with moist sounds and whispering bronchophony. Tubular breathing also at right base, with crepitation all down the left side. *Oct.* 25.—Respirations laboured, 35; pulse 140, very small and feeble. Profuse perspirations; friction sounds over right base. Died same day.	*Heart* sounds clear and sharp; pulse ... ...sonance markedly increased over right base.	*Right lung.*—Signs of recent pleurisy, but no firm adhesions. On section, upper lobe of dark red colour, and interlobular tissue appearing increased. Very small quantity of fluid from surface of section, and tissue not breaking down under finger. The whole of lower half of right lung solid, firm, and somewhat tough; of reddish-grey colour, and offering some amount of resistance to the knife. Sank in water, and exuded scarcely any fluid when pressed. *Left lung* in a similar condition, except that the consolidation was arranged more in patches. *Bronchial tubes* much congested, but not dilated. *Bronchial glands* much enlarged. *Heart* healthy, except for contraction and puckering of one of columnæ carneæ. *Liver*, normal. *Spleen*, very large and firm. *Kidneys*, large, very firm and tough. *Intestines*, healthy.
III. M. æt. 61, M. Charcot, De la Pneumonie Chronique, Thèse de Paris.	A hosier; delicate looking; generally enjoyed good health, but has had a cough for some months, and has grown rather thin.	SYMPTOMS, &c. (continued) right lung, with, at first, simple febrile, and afterwards typhoid, symptoms. *April* 4.—General condition improved; ... instead of nasty...	SYMPTOMS, &c. (continued) Loud, not even bronchial breathing. No ægophony. *April* 18.—Local signs continued of approaching change; but return of appetite; perspirations at night, and	*Right lung*, universally adherent by old and tough adhesions; pleura constituting a fibrous envelope ¼" in thickness. Whole organ ⅓ smaller than left lung. Tissue heavy, dense; finger cannot penetrate it; on section, resisted scalpel like fibro-cartilage... The three lobes were seen to be...

		...or night sweats. July 1–5.—Gradually became worse; the hectic persisting, but still none of legs. On the 9th, the expectoration (being before scanty) became very abundant and somewhat mummulated. On this day the following *Physical Signs* were recorded. On *left* side, resonance good, with puerile respirations. On *right* side, below clavicle, marked dulness; respiratory murmur faint and indis-	...with large metallic râles, simulating gargouillement. Bronchophony, but not pectoriloquy. But not always the same result; sometimes *complete silence* over whole of upper and lower lobes, and sometimes *tubular breathing mixed with large metallic râles*. No note made as to variations in expectoration at these times. Died on *July 19th*, much emaciated.	...mating the tongue swollen, and found to be healthy. A flatulent opening existed near the lower angle of right scapula, leading into the cavity of a large old abscess over the third, fourth, fifth, and sixth ribs, whose external surface was in a carious and necrosed condition, whilst the intercostal muscles were partly destroyed. The internal surface of these ribs was quite healthy, and no communication existed between the cavity of the abscess and that of the chest.
IV. M. æt. 30. Dr. J. E. Pollock.	A soldier in India for 5½ years, and afterwards a warehouse porter. Tall, well-made, and pretty well nourished, though he had lost flesh. General health good.	*May 8.*—Lower angle of right scapula raised by a *large and deep abscess*, from which, on incision, issued about 15 oz. of pus. Up to *June 1st* the abscess continued to discharge, though fluid gradually more serous in nature. During this time there were hectic fever and rapid wasting, but no diarrhœa. All the signs of pulmonary indium-	Suffering from cough for the last nine months, and streaky *hæmoptysis* for six months. Expectoration profuse, frothy, and muco-purulent. Bowels constipated. Occasional pain in back between the shoulders. Death from rupture of aortic aneurism into left bronchus.	Heart's impulse visible from apex region to second left cartilage. General contraction of whole left side, with flattening in front, and slight depression of shoulder. Dulness not absolute over *left* side; respiration very deficient in subclavicular region, with increased vocal resonance over apex posteriorly. On *right* side, increased resonance up to and beyond left of sternum. Respiration normal.	*Left lung*, adherent throughout, contracted; could only be removed together with costal pleura. On section, bronchi greatly dilated, with intervening tissue fibroid and airless. *Right lung* enlarged, covering pericardium. *Heart* healthy; aortic valves thickened, but not incompetent. *Aneurism* of descending part of aortic arch, ruptured.
V. M. æt. 7½. (M. Legendre.) Barth., in Mém. de la Soc. Méd. d'Observat. de Paris, 1856, t. iii.	Parents healthy, was brought up by hand, took violent cold a few days after birth. Three weeks after began to vomit food, which continued for a long time. Cut teeth and walked at usual time. When 3½ or 4 years old, began to expectorate large quantities of purulent matter. Two or three times a day at most, after feeling of anxiety and face becoming red, there were paroxysms of cough, with copious ejections of pus. Felt relieved immediately	*Oct. 11, 1841.*—In morning, skin cool, pale; pulse 80; but about 5 o'clock face becomes red, skin hot, pulse 116–120, respiration frequent, and in night abundant sweats. Two or three hours after meal, at 11 A.M., feeling of malaise with anxiety, and in fifteen or twenty minutes the cough comes with floods of expectoration, and often vomiting of food. Fluid thin, purulent, with stale colour; from 6 to 8 oz., though less when night attack also, as often. *Dec. 13.*—Has lost flesh lately; evening attack constant, night sweats copious.	Tongue moist, belly big, with tenderness over region of enlarged spleen. *Right* side resonant all over. On *left*, under clavicle, as good as right side, but behind completely dull from top to bottom. On right side, and under left clavicle, respiratory murmur normal; but over whole of left back, respiration cavernous, with râles, and also great vocal resonance. *Dec. 31.*—Not having been auscultated for several days, in addition to previous signs, there was detected under left clavicle a slight dulness, with bronchial breathing and large mucous râles.	Pleural adhesions very slight, principally on left side. *Left lung* not diminished in size. Lower lobe heavy, hard; no appearance of air-vesicles on section, but seen to be converted into a dense, reddish, homogeneous tissue, containing cavities from size of pea to almond filled with expectoration matter. This part—in colour, consistence, and density—was like tissue of an enlarged uterus. Bronchi normal, as far as first division; but afterwards, *uniformly* dilated throughout. Upper lobe nearly as heavy as lower, but not nearly so dense; and consistent, though airless. Tissue reddish-grey, granular on surface of section, but breaks down less easily than

Sex, Age, and Observer.	General History.	Symptoms.	Inspection, Percussion, Auscultation, etc.	Autopsy.
V. (continued).	after this. In intervals neither coughed nor spat. Habitual dyspnœa; skin hot at night, and copious sweats, but no diarrhœa; and appetite always good. From this time to 7th year continued much the same, but grew and was by no means thin. Of moderate size and fatness; skin pale; skin thick; intelligence good. No injection of face.	*Dec.* 31.—Thinner; bad diarrhœa for two days. *Jan.* 12.—Feebler and thinner, much wasted, slight bed-sores. Breath fetid; ulceration of gums and inner side of right cheek. Appetite less; still diarrhœa. *Jan.* 14.—Gangrene of gums and inside of right cheek progressing, and breath very fetid. *Jan.* 16.—Died		recent hepatization. Bronchi only slightly dilated. Not the least trace of tubercle or grey granulation. *Right lung*, healthy and crepitant, except for a few "grey granulations" scattered through its substance. *Bronchial glands* on left side enlarged, reddish grey. *Kidneys and liver* healthy. *Spleen* large and very consistent. Mucous membrane of intestine healthy.
VI. M. æt. 34. Dr. Barlow. *Path. Trans.* vol. xvi. p. 29. Univ. of Lond. Cal., 1867, p. clxvii.	A strong, powerful-looking man. A sailor, and then a labourer; of very intemperate habits. Nine years ago had an attack of hæmoptysis (after a fall) which continued for some weeks; then remained well till four or five months ago; when felt severe pains in left back, had dyspnœa, cough, and *hæmoptysis*; also grew thinner. For three weeks had been laid up, and his feet had swollen. Never had rheumatism.	Great dyspnœa and violent cough, respiration only 32. Skin moist, tongue slightly furred; pulse 72, regular, full, but soft. Appetite good. Lies on left side. Expectoration purulent, tenacious, and mixed with blood. Urine scanty, depositing dark lithates. After about three weeks he became much worse, the dyspnœa and lividity increasing, and died in ten days' time from the supervention of an attack of acute bronchitis.	[face and lips] usual, but livid on coughing. Veins of neck turgid and pulsatile. Ankles œdematous, and a few spots of purpura on arms. *Heart's* beat seen at the epigastrium, but apex felt in usual position. Signs of loud tricuspid regurgitant murmur. *Left side* of chest appeared somewhat contracted, and much less moveable. Extremely dull, but chiefly at upper part. A faint respiratory murmur heard at base; but above, sounds bronchial and cavernous, with mucous rales. Bronchophony at apex. *Right side.*—Respiration puerile all over.	*Left pleura* enormously thickened, and almost whole of lung converted into a gristly mass, which, on section, showed the cut ends of the great vessels and the shrivelled tubes, with a great deal of white fibroid tissue; only a few islets of healthy tissue here and there. Mucous membrane of bronchi red and thickened; the secondary bronchi, and all the smaller branches, were shrunken and very red within, but there were *no dilatations*. *Right lung* very large, but tissue healthy. *Bronchial glands* healthy. *Heart* very large; left ventricle healthy, but right ventricle dilated and thickened, and right auricle in same condition. Tricuspid valve thickened. *Liver* large, having "nutmeg" appearance. *Kidneys* large, coarse, and congested.
VII. M. æt. 34. Dr. Mayne. Dublin Hosp. [...]	A labourer. Strong, and of robust constitution. Never had a pain or ache till July 1855, when, after hard day's [...]	On admission, no hectic; pulse only 80; scarcely any sweating; no diarrhœa. Expectoration at first [...]	No displacement of heart, or difference in attempt two sides of chest; but diminished mobility of right [...]	*Right lung* universally adherent. Adhesions loose above, but very dense below, and inseparable from diaphragm. Lobes closely united. [...]

			...in emphysema, with a corresponding odour. *Left lung* perfectly healthy. *Heart and abdominal organs* healthy.
VIII. F. æt. 3?. Dr. Bright, Miscellan. Inspection, Book 19; and Mus. Prep. No. 1718* (Guy's Hosp.).	Ailing from infancy, but for last two years increasing cough and weakness.	Perspires profusely at night; sleeps well, and generally drowsy. Expectoration profuse — yellow clots in thin fluid. Pulse 120, small. Diarrhœa.	Body much emaciated. Heart sounds and impulse most distinct between second and third right cartilages. Flattening of right side, chiefly in sub-clavicular region. *Right side* dull anteriorly, except just below clavicle. Tympanitic resonance in inferior lateral region. Behind, resonance fair. On auscultation, gurgling with metallic resonance and voice ringing. *Left side.*—Increased resonance, all over. Respiration puerile.	*Right lung* firmly adherent above, but more loosely in lower parts. Upper fourth of lung dense, pale, and, but for some of large tubes, devoid of air. Inferiorly and posteriorly was an extensive series of cavities—dilated tubes—between size of fowl's egg and that of sparrow; some containing a semi-solid mucous secretion. *Left lung* uniformly "fleshy, red, lacerable, and watery." *Heart* smallish. *Liver* pale and oily. *Kidneys* light, slightly granular, and somewhat firm.
IX. M. æt. 29. Dr. Foot, Dublin Quart. Journal, Feb. 1866, p. 200.	Pulmonary symptoms dating from an attack of bronchitis two years ago. No profuse *hæmoptysis* previous to one which proved fatal.	Expectoration occasionally streaked with blood.	*Left side* comparatively flattened.	*Left lung* small and retracted deeply into chest, drawing heart with it; adherent to ribs and diaphragm—had to be cut out. Substance firm, leathery, non-crepitant, and sank in water. On section, showed dilatation of bronchial tubes, and in its apex was a cavity, the size of an acorn, full of blood. *Right lung* large, congested, and emphysematous, extending into left side of thorax, and containing in its apex a "tubercular cavity," of the size of a walnut, which was filled with a black coagulum. Blood also in bronchi, trachea, pharynx, and stomach.
X. M. æt. 40. Dr. Dickinson, Path. Trans., vol. xiii. p. 60.	A labourer. Had been ill two years, with cough, and pain on right side of chest.	On admission, wasted and feeble, with much cough, dyspnœa and frothy expectoration. Urine albuminous. One month later, increasing ascites with œdema of legs; cough still frequent. Strength then failed, dyspnœa increased with ascites; and patient died in another month.	Ascites and anasarca. Basic systolic cardiac murmur. *Right side* flattened in front. Breath sounds very deficient over whole side. Behind, dulness on percussion, with increased vocal sound. Over whole of *left side* breath sounds exaggerated.	*Right lung* firmly adherent at every part to parietes, small and solid, sinking in water. Pleura and interlobular septa greatly thickened. Bronchial tubes natural. Pulmonary artery contracted, and containing a fibrinous clot. *Left lung* healthy, though somewhat compressed by dilated pericardium. *Pericardium* distended with bloody fluid, walls thickened and covered with old lymph.

Sex, Age, and Observer.	General History.	Symptoms.	Inspection, Percussion, Auscultation, etc.	Autopsy.
X. *(continued).*				*Heart* fatty (?); continuous fibrinous matter in right side, and right pulmonary artery adherent to muscular wall of ventricle. *Liver* very large, waxy, and cirrhosed.
XI. M. æt. 55. M. Jaccoud, Clinique Médicale. Paris, 1867, p. 83.	Till three years since quite healthy; then began to cough. Has had *hæmoptysis*, has grown weak, and has become thin. Breath more and more short. One year ago was in hospital for acute chest symptoms (pleurisy), and remained two and a half months. Left, free from acute, and, for a time also, better as regards chronic symptoms. But these returned in a few weeks. Expectoration became more abundant in morning.—" un véritable vomique." Feebleness and emaciation increased ; legs began to swell, though patient was almost entirely confined to bed.	Looks old & pale, and very much emaciated. Tormented with paroxysms of cough. Continual dyspnœa; feebleness considerable ; no fever. Œdetus as far as knees. Abundant purulent homogeneous sputa, without mixture of mucosity. Gradually got worse ; wasting continued ; no diarrhœa, but enormous expectoration ; also fever at night, terminating in abundant sweats. Earthy and wan look of face, with great general emaciation. Four weeks afterwards, without any notable alteration in the symptoms or physical signs, the patient died.	Heart sounds normal. No flattening of chest noted. On *right* side of chest absolute dulness anteriorly, with great resistance to finger. Same in lower two-thirds of right back, but less marked in upper one-third. Vocal vibrations much increased over the whole of dull area. In front, under right clavicle, large râles and bronchial breathing ; also exaggerated resonance of voice and cough. Râles larger and larger on descending ; and at level of nipple so large as to resemble gargouillement together with cavernous breathing and pectriloquy. Below line of nipple, again, intense bronchial breathing, with marked bronchophony. But in centre of this portion are large moist râles in two very limited spaces. Same signs as at nipple level. Posteriorly, very exaggerated respiration above, with snuckling on cough or strong inspiration. At level of lower angle of scapula, much the same sounds as at nipple level. Quite at base, posteriorly and laterally, intense pleural friction sounds. On *left* side, percussion perfectly clear, and breath sounds healthy.	*Right lung* indurated, of fibro-cartilaginous consistence ; contained a very small portion of crude and slightly softened "tubercle" at the posterior part of apex. No cavities due to softening of "tubercle," but many to dilatation of the bronchi—the largest being opposite mammary region. Lung-tissue presented a good type of general sclerosis. For a depth of three fingers' breadth the inferior lobe was united to the parietes by old, false membranes, which were sufficiently loose to permit of rhythmical displacement of the organ. *Left lung* healthy.
XII. F. æt. 16.	Attacks of bronchitis and pneumonia on several occa-	In last attack there was extreme dyspnœa, with universal crepitation	Impulse of heart felt under left clavicle.	*Left lung* a mere rudiment, forming a small mass of tough, fibro-cellular substance of atelectasis, when cut into, the mouths of

signs of phthisis.

XIII. M. æt. 62. Dr. Addison, Clinical Ward Book, Oct. 1859, and P. M. Records, 1859, No. 293.	An engine-driver. Emaciated, but with appearance of once having been well-nourished. Exposed to great alternations of heat and cold. Was in good health till four or five years since. Had typhus fever three years ago. Since then has felt slight pain and sense of weight in chest.	Cough and expectoration. Pulse 60, feeble; tongue clean; bowels regular; urine not albuminous. After three weeks, urine contained a little albumen. The next week he expectorated a small quantity of blood; and in another week he died, after increased weakness and asphyxial symptoms.	Legs anasarcous, with slight general oedema. Considerable lateral curvature, convexity of chest to right (attributed to occupation). Heart apparently enlarged, with a tricuspid regurgitant bruit. Pulsation in jugulars. Right side abnormally dull posteriorly; bronchial rales with prolonged expiration in upper half, with crepitation over lower half of lung. Bronchophony over upper lobe. Left lung resonant; bronchial rales all over.	(margin projecting into left side of thorax), "with a few tubercles scattered through it." Heart immediately under left clavicle. Right lung universally adherent by tough tissue—cut out. Organ consolidated from end to end; only a few small portions permeable to air. Tissue dense, tough, and fibrous; colour blackish. Apparently several centres of induration. No softening, except at apex, where a very small cavity. Tubes not much affected; in upper part rather large and thickened, but elsewhere unaltered. Pulmonary artery throughout much dilated; coats much thickened, and lined throughout with atheromatous deposit. Some of smaller branches of artery, and also of pulmonary veins, obstructed by ante-mortem coagula. In main pulmonary artery a closely-adherent layer of fibrine. Left lung also showed some slighter induration; large bronchi intensely red, and filled with mucus. Heart.—Hypertrophy, with dilatation of right side. Tricuspid valve thickened. Liver.—Early stage of "nutmeg" appearance. Kidneys.—Large, and slightly granular on surface.
XIV. F. æt. 24. Dr. Corrigan, Dublin Hosp. Gaz. Dec. 1857.	A bookbinder. Healthy and strong up to four years ago, when slept in damp clothes. This induced cold and cough, with burning pain in right shoulder. The cough abated, but pain continued. Health improved in summer, but worse in winter—with occasional oedema of the feet.	Debility, cough, diarrhœa. Not so much emaciated as might have been expected. From invalid condition, rapidly sank and wasted, owing to uncontrollable diarrhœa, which lasted nearly six weeks.	Heart's impulse 2° to right of sternum and above level of third rib. Whole right side of chest dull, with bronchial or cavernous respiration. Left side, including cardiac region, clear on percussion, with exception of superior portion, which dull with cavernous respiration.	Right lung less than half natural size; middle lobe quite rudimentary. On section, all three lobes show great number of dilated bronchial tubes, lined by a smooth glistening membrane. Intermediate tissue grey, tough, non-crepitant, and apparently devoid of blood. Finer bronchial tubes obliterated; larger, showing circular fibres within drawn apart by contracting tissue. One small speck of tubercle found. Universal adhesion of both pleural surfaces. Intestine. Ulceration of cæcum and ilio-cæcal valve.

SEX, AGE, AND OBSERVER.	GENERAL HISTORY.	SYMPTOMS.	INSPECTION, PERCUSSION, AUSCULTATION, ETC.	AUTOPSY.
XV. M. æt. 46. M. Charcot. PneumonieChronique, Paris, 1860, p. 19.	A powerfully-made man. Had always enjoyed good health till four years ago, when he had a chest affection, for which he remained in hospital one month. Never properly recovered, and since then has become weak and cachectic—only able to work occasionally; with colds in winter. Has spat pure *blood* several times. Two or three months ago, when again in hospital, brought up *blood* copiously.	Entered hospital again on *July 16th*, having been much worse for last eight days. Was very thin; skin cool, pulse frequent, and complained indefinitely of pain on right side. Sputa copious, muco-purulent, green, not fetid. No shivering, but complete anorexia. Tongue with thick white fur. In eight days' time was restored to original condition. On July 30th, and subsequent eight days, sputa more copious and of *gangrenous* odour. Also became weak, thin, and had uncontrollable serous diarrhœa. After Aug. 5th, better; no fœtor nor diarrhœa; slight œdema of legs, and soon after decided anasarca. Physical signs the same. Became weaker, with hectic at night, till *Sept. 25th.* Sputa again very abundant, with trace of blood, but not fetid. Uncontrollable choleraic diarrhœa. Extremities cool; skin violet; pulse small, weak. Died on Oct. 2d.	*Heart* normal. Slightly exaggerated resonance in both infra-scapular regions. Respiratory sounds there dull, but no râles or crackling. Behind, same characters. *Right* side, marked dulness from point of scapula to liver, with great resistance; with deep, dull, tympanitic sound below. Over dull part, distinct bronchial breathing, with large râles; and over region of tympanitic sounds cavernous souffle, with gargouillement. Bronchophony over whole lower lobe.	On opening thorax, both lungs pale and emphysematous above, with no adhesions, except around *lower lobe of right lung.* This was reduced to half of natural size, and was universally adherent to diaphragm and vertebræ. On section, it cut like fibro-cartilage, and presented a smooth surface of deep slate-colour, intersected by fibrous bands. Bronchial tubes not dilated, lined by a vividly-red membrane. At posterior border, near surface, was an elongated excavation, having irregular walls, but pretty smooth surface of lung tissue, containing greenish serous pus of stale odour. Cavity divided here and there by bronchi and bands of tissue, and large bronchi open into it. In front of this, and near diaphragm, was a softened patch of tissue (size of franc-piece), yellowish, with strong, but not gangrenous, odour. *Heart, liver,* and *kidneys,* healthy. *No tubercle* in either lung. *Ilium* and *colon,* mucous membrane of, violet-coloured, with white tenacious mucus.
XVI. M. æt. 28. Dr. Wilks. Path. Trans., vol. viii. p. 39.	A hairdresser, of dissolute habits, who had drunk freely of gin. Four years ago was ill in St. Thomas's Hospital with a cough. Not well since, through able to follow occupation. No extreme emaciation.	Symptoms severe for ten weeks before death. Suffering from pain in chest, extreme dyspnœa, much cough; little expectoration, but occasional *hæmoptysis.* Unable to lie on right side. Latterly, increased dyspnœa and lividity, with purpuric spots on legs.	Face and body livid; ascites and anasarca. Heart considerably further to left than natural. Systolic bruit (?). *Left side* of chest contracted and almost immoveable, with dull percussion note, and cavernous respiration.	*Left lung* had to be cut out; much reduced in size. Pleura, in some places, (?' in thickness, and of fibro-cartilaginous hardness, continuous (on section) with white smooth bands, intersecting lung between dilated bronchial tubes — the latter occupying half the bulk of lung. All the tubes altered, especially those of lower lobe. Healthy mucous membrane gone, and in many places they were more membranous canals terminating inferiorly in large round cavities with same kind of lining.

XVII. M. æt. 19. Dr. Peacock, Month. Journ. of Med., April 1855, p. 281.	When seen in Nov. 1849, had been ailing for three years, with cough and expectoration. General health impaired. No pain in chest; much emaciated; and face somewhat livid. Improved under ol. morrhuæ and a generous diet. Pulse 100, regular; tongue clean; appetite moderate.	Expectoration at first very profuse, fœtid, brownish-coloured, and purulent. Afterwards less profuse, but brought up in gulps; at other times pretty free from cough. In beginning of April 1851, after exposure to cold, was seized with great dyspnœa, increased cough, and considerable *hæmoptysis*. Died asphyxiated on 11th of April—4½ years from commencement of illness.	*Right side* flattened in front, especially between outer end of clavicle and nipple. Heart's impulse seen below right nipple. Resonance impaired in right sub-clavicular region, and complete dulness below nipple. Vesicular murmur heard only immediately below inner half of clavicle; below outer extremity of same, loud gurgling rales, and cavernous sounds with voice and cough; resonance tympanitic. *Left side.* Percussion sounds clear throughout, with loud and puerile respiration.	*Right lung* very small; almost entirely adherent by old attachments; at base, adhesions which seemed more recent. On section, everywhere consolidated, except at upper and anterior edge; condensed portions very firm, pale fawn colour, mottled with black pigment. Primary and secondary bronchi dilated, and opening abruptly into cavities of various sizes—some, towards apex, oval with smooth surfaces continuous with membrane of tubes; others, not this appearance, but that of old ulcerated cavities communicating with several bronchial tubes. In lower lobe, a recently-ulcerated cavity, containing a considerable quantity of coagulum, communicating with bronchi whose mucous membrane was vascular and ulcerated here and there. Bronchial tubes in upper part of lung nearly free from secretion; but those of middle and lower parts contained muco-purulent matter deeply tinged with blood. *Left lung* very large, extending far to right side, and completely hiding right lung.
XVIII. M. æt. 33. Dr. Sibson and Dr. Bastian, Path. Trans. vol. xix. p. 44.	A boatman, not much emaciated, who had suffered from cough for years, but had been comparatively well till one week before death.	A sudden chill followed by great dyspnœa and headache. Expectoration sticky and mucoid. Pulse quick and feeble, 100. Respiration 36, lying on back.	Face and lips purple. Flattening of *left* side of chest most notable under clavicle. Dulness under clavicle and whole of left front; and, in same situation, mucous and coarse crepitant rales. Percussion on right side, normal.	*Left lung* had to be cut out around apex and posterior parts. Organ much contracted. Pleura in some parts more than ¾" thick, and of cartilaginous consistence. Fibroid conversion of lung-tissue, with trabeculæ extending inwards for depth of 1" throughout upper lobe. Immediately beneath this, a smooth-walled cavity, and one with ulcerated walls—both having purulent contents. Some bronchi of upper lobe uniformly dilated. Lower lobe much less altered; tissue firm, but œdematous. *Right lung* large, congested, and œdematous throughout, but chiefly so in upper lobe. *Heart.*—Right ventricle enlarged and auricle much distended with blood. Left side, normal. *Liver, spleen,* and *kidneys*, healthy.

Sex, Age, and Observer.	General History.	Symptoms.	Inspection, Percussion, Auscultation, etc.	Autopsy.
XIX. F. æt. 30. Dr. Corrigan. Dublin Journ. of Med. 1838, p. 273.	Had been for some years complaining of her chest, and unable to lie on her left side.	Serious symptoms only for last six months, cough, and palpitation of the heart came on. When seen, there was great dyspnœa and very troublesome cough, with a viscid and purulent expectoration. Pulse 100, weak. Thirst, vomiting, and diarrhœa.	Ascites and anasarca, but no lividity of face. *Heart's* impulse under right mamma. *Right* side remarkably dull throughout. Antero-superiorly, gargouillement, with cavernous respiration and pectoriloquy. Posteriorly, similar signs, but not so distinct. *Left side* clear on percussion, but least so antero-superiorly, where there was a loud mucous rattle and some vocal resonance.	*Right lung* size of two fists, in upper part of thorax, and intimately adherent by fine cellular tissue. On section, the lung presented a "confused congeries of large bronchial tubes united by dense, firm tissue." No trace of healthy structure or of tubercular cavities. Bronchial tubes ending abruptly in rounded cavities filled with muco-purulent matter. Remainder of right thorax occupied by heart and liver, which latter was unusually high. *Left lung* enlarged by at least one-half, contained four or five small abscesses in its centre; and in the lower lobe, some small portions in a state of red and grey hepatization.
XX. M. æt. 36. Dr. Greene. Dublin Quart. Journ. 1846, p. 510.	Five years ago fell on chest, after which suffered from dyspnœa.	Six months ago felt pain in side, cough, and difficult respiration. On admission, slightly jaundiced, and much emaciated; with cough, and expectoration small in quantity and fœtid. On second day, had fever; and on third day, delirium; and on fourth, died.	Great prominence in hepatic region, with sulcus between liver and margin of chest. No pulsation in precordial region. *Right side* of chest contracted and immoveable. Dull on percussion, with bronchial respiration, bronchophony, and great vocal resonance. No vocal fremitus; mucous rales all over. *Left side,* clear. Respiration puerile, —extending to right side.	On opening thorax, *right lung* at first not seen, being at back part with heart anterior to it in mammary region, apex pointing downwards and to left. This lung adherent to pericardium, and when cut into found to be solidified and the bronchial tubes "larger than natural." *Left lung* very large, extending under sternum to right.
XXI. M. æt. 40. Dr. Jennings. Dublin Quart. Journ. Feb. 1866, p. 205.	A bricklayer, much exposed to wet and cold. Five years ago caught severe cold, attended with profuse hæmoptysis, which continued for three days, and in less degree for some time afterwards. Cough and expectoration.	Incessant cough, and copious expectoration of yellow, viscid, muco-purulent matter. Skin cool. Able to lie only on right side. Pulse 80, regular.	*Heart's* apex impinging below *right* nipple. Shrinking of *right* side of chest, with flattening of sub-clavicular region. Right shoulder lower than left. General dulness of same side, and marked immobility. Under right clavicle, ...	*Right lung* a small, hard, tough mass of fibro-cartilaginous consistence. On section, seen to be traversed by widely-dilated bronchial tubes, ending near surface in cavities of considerable size (large nut), lined by a "dense, whitish membrane." Radiating lines of fibrous tissue very evident.

	Two years ago dropsy supervened. Eighteen months after, again seen, when profuse muco-purulent expectoration, lips livid, jugulars distended, and great dyspnœa—much increased by even slight exertion. Not much emaciation or hectic.	shortly after admission.	...clear on percussion, but dulness over lower three-fourths. No vesicular murmur over right lung, except in infra-clavicular space, whilst posteriorly, below spine of scapula, were heard bronchophony, tubular breathing, and mucous rales. Immediately above and below the spine of scapula, pectoriloquy and gurgling rales. Over *left side* (including cardiac region) percussion sounds clear.	*Left lung* increased in size, congested, and œdematous. *Heart* to right of sternum, with apex directed towards right side. Left cavities, normal; but those of right dilated and unusually hypertrophied. Right auriculo-ventricular opening much dilated. Trunk of right pulmonary artery filled with firm, laminated, colourless fibrine, adhering to wall of vessel. Left branch larger than natural, and unobstructed.
XXIII. M. æt. 20. Dr. Mayne. Dublin Hosp. Gaz 1860, p. 33.	Inhabitant of a Union. Six years ago was admitted into the hospital of the Union for what appeared to be phthisis—cough, loss of flesh and strength. (No early stethoscopic signs recorded.) Cough, expectoration, and dyspnœa continued for three years, though strength improved. During an interval of 2½ years he travelled about the world and underwent an extreme amount of exertion and fatigue. When seen five months ago, had so much improved in appearance as to be scarcely recognisable.	*Five months before death.*—Appetite very good; bowels rather costive; pulse 80; seldom sweated, and never profusely. Colour good; condition moderately plump. Breath habitually short, but this caused no distress unless heart's action was hurried by exertion. Cough; and free expectoration of muco-purulent matter, especially in the *morning.* *One week* before death, caught cold—severe bronchitis affecting whole of sound lung, with rales over every inch of it. Fever of unfavourable type; extremely rapid pulse. Got gradually worse, and on third day suffocation seemed imminent. Habitual orthopnœa; lips purple. Fourth day delirious (insufficient aëration of blood). On fifth day, comatose; and on seventh, died.	*Five months before death.* Pulsation of *Heart* was seen and felt beneath left axilla; the sounds and rhythm being unaltered. *Left side* contracted to extraordinary degree, vertically and horizontally; intercostal spaces narrowed; immobility of ribs. Left shoulder depressed, with habitual stoop to left side. *Right side* full and plump; intercostal spaces wide; great mobility of ribs. *Left side,* on percussion, dull throughout. On auscultation, at upper and posterior portions, nearly all the physical signs of a large cavity communicating with a bronchus. In other portions, mucous rales in every variety; also bronchial breathing and voice-sound at intervals. *Right side,* on percussion, almost tympanitic, beyond natural limits laterally and inferiorly. Breath sounds, on this side, exaggerated.	*Left lung* scarcely size of hand, firmly adherent posteriorly, and to pericardium, which, with heart, was drawn up. Substance dense, tough, and resisting, and capped superiorly and posteriorly by a dense fibro-cartilaginous layer, which had to be cut out. On section of thick, posterior edge of lung, in its upper part was a large cavity ($4'' \times 2\frac{1}{2}''$), having irregular walls, dense and of buff colour; crossing cavity was a band of tissue, and on walls the patent orifices of several bronchial tubes. Tissue around dense and fibrous; contained a few pea-like, chalky masses, but no recent tubercle. Other parts of lung showed same dense fibrous tissue, throughout which bronchial tubes were traceable almost to pleura; these were considerably enlarged and widened, but did not present the enormous dilatations and *culs de sac* which are sometimes met with. *Right lung* enlarged and hypertrophied, extending beyond the middle line of sternum, and also lower than usual.

Sex, Age, and Observer.	General History.	Symptoms.	Inspection, Percussion, Auscultation, etc.	Autopsy.
XXIV. M. æt. 63. Dr. Walshe. Medical Times, Feb. 16, 1856, p. 157.	A labourer. Had lived in dry, comfortable houses; never had rheumatism or syphilis. For many years a sufferer from cough, but much worse for ten weeks before admission, when he left off work. Sleep usually good, now disturbed. No night sweats. Pain behind sternum.	Jan. 19, 1855.—Slept on right side; face flushed; bowels confined; respiration 32, steady; no subjective dyspnœa; pulse 112, regular, small. Trifling aching at left mamma. Cough frequent; sputa copious, running into one muco-purulent mass. After one month, diarrhœa for a week or two; and when this ceased, was weaker and more emaciated. Then became better again, and continued so for more than a month. On April 21st, skin hot; face flushed; feverish; no pain; expired air very fœtid; and fine moist rales at left inferior back. On April 28th, expectoration watery, purulent, and very fœtid. Gradually sank, and died on April 30th.	Heart's impulse felt on right side; sounds natural. Scarcely any appreciable shrinking of right side, but less mobility. Right supra-spinous fossa strikingly sunken; and percussion here tubular, as well as in axilla. All over right back, wooden percussion-note. Whispered pectoriloquy in right supra-scapular fossa. Under right clavicle, percussion high-pitched, tubular; and here respiration bronchial, with strong diffused bronchophony. Heart sounds also very loud. Vocal fremitus less at right than left posterior base. Under left clavicle, extra resonant, with exaggerated breath sounds. Over left back also extra resonant, with respiration loud and hollow.	Right lung most closely adherent to pericardium, diaphragm, and side; forms bed on which lie heart and great vessels. On section, creaks under scalpel; tissue dense, whitish—this most marked at surface for depth of ⅜", in parts. Pulmonary vessels increased in size, and also dilatation of the bronchi—main bronchus and secondary—in irregular manner. Walls deep red, and thickened. At lower anterior part of lung, a cavern of doubtful character, size of hazel-nut, filled with pus. Bronchial glands cirrhosed. Left lung much enlarged, extending into right side of thorax. The greater part easily torn, much infiltrated with frothy serum, and exhaling same fœtid odour as sputa during life. Some firmish lobules infiltrated with greyish-red material, but no tubercle. Upper part of upper lobe healthy. Heart, hypertrophy and dilatation of right auricle and ventricle. Left ventricle smaller than natural, fatty, and with fibroid conversion of one papillary muscle. Kidneys coarse, cortical substance narrow, tissue hard.
XXV. M. æt. 29. Dr. Gull. Clinical Case Book, 1865, & Post-mort. Records, No. 83. (Guy's Hosp.)	Dock labourer and potman. Fourteen years ago, in Guy's Hospital for a chest affection, producing orthopnœa. Delicate ever since. Whilst at work, often several hours up to waist in water. Ten months ago suddenly felt a gnawing pain under left nipple, extending to scapula. Three weeks ago his abdomen be...	On admission, March 8th, great dyspnœa, and occasional cough, with expectoration of purulent mucus—not tenacious, but streaked with blood. Pain in scrobiculis cordis and in left lumbar region. Bowels open; urine very scanty. Afterwards complained of more pain over abdomen. Great tenesmus and loose stools for two days; then took stools cold on night of 20th, grew worse till over, ...	Abdomen swollen and tense; legs œdematous; but above waist body somewhat emaciated. Superficial veins of abdomen injected, filling from below. A pulsating vein dipped down between 2nd and second ribs. Heart drawn up; apex beat just over left nipple, but impulse of entire ventricle felt. Sounds normal. Dulness over area ... whole of left side ...	Left lung very much diminished; adherent to parietes by such dense fibrous tissue that it had to be cut out. Pleura of great thickness and cartilaginous hardness, especially that of upper lobe and between lobes; lung-substance within, forming a hard, grey, fibrous mass. Lower lobe still spongy, but hard and containing very little air. The bronchi in upper part were slightly dilated; their mucous membrane being dark-red, and velvety. The ...

				Chronic peritonitis, with ...... thickening of its capsule, and liver; great thickening of its capsule, and adhesion to adjacent parts. Kidneys and Spleen healthy.
XXVI. M. æt. 40. Dr. Stokes, of Diseases of Chest, 1837, p. 157.	Asthmatic cough since boyhood.	Chronic bronchitis, with paroxysms of orthopnœa. Copious expectoration of masses of yellow colour, which flow together. Lies on left side.	*Heart's* impulse strong, with violent pulsation at epigastrium. *Left* side of chest not altered, but the *right* singularly convex from third to seventh rib. Postero-inferior portion of *left* side dull, with vocal resonance almost amounting to pectoriloquy. Right side morbidly clear; but respiratory murmur generally feeble and almost replaced by a sibilant rale.	*Left lung* firmly and universally adherent; very small. Lobes united by recent lymph; upper covered with large emphysematous vesicles; lower, pale, of flabby consistence, but also showing dilated air-cells. On section, a large cavity—size of apple, evidently a dilated bronchus—containing a viscid, yellow fluid. All bronchial tubes on this side more or less affected, so that lung appeared to contain many small abscesses. Posteriorly, pulmonary tissue dark grey and of cartilaginous hardness. *Right lung* twice as large as left, and very emphysematous. Tubes bright red, and filled with muco-purulent fluid. Universally adherent. *Heart* twice natural size; right cavities dilated and hypertrophied. Left ventricle, hypertrophied only. Valves and aorta healthy.
XXVII. F. æt. 39. Hérard and Cornil, De la Phthisie Pulmonaire, p. 168.	A cook. Has had habitual cough, especially in winter. Increased weakness during the last twelve months. Most marked during last month.	Considerable *hæmoptysis* twelve months ago. Now, great dyspnœa, with lividity of lips and cheeks. Sputa purulent, slightly aërated, and almost nummulated. Thirty hours before death, fell, paralysed on left side, with coma and stertor.	Slight ascites and anasarca. *Heart* sounds, normal; pulse, small and frequent. Sphygmographic tracing similar to that in asphyxia. On *left side*, large cavernous rales. Whole of *right side* hyper-resonant, with fine rales and sibilant respiration towards summit.	*Left lung* small, globular—adherent to vertebral column and to summit of thorax. Lobes united, and anterior border of lower presenting emphysematous dilatations the size of a nut. Superior lobe containing two large cavities (size of egg) with smooth lining, continuous with mucous membrane of larger bronchi, which congested and covered with muco-pus. Surrounded by dense tissue, continuous with thickened pleura, 2″ in depth. *Right lung* enormous, with large emphysematous bullæ at borders. *Heart* large. Left cavities, normal; right, dilated, with thickened and stiffened walls. Fluid in pericardium and in peritonæum. *Liver* granular on surface and very hard. *Spleen* large and hard.

Sex, Age, and Observer.	General History.	Symptoms.	Inspection, Percussion, Auscultation, etc.	Autopsy.
XXVIII. M. Laennec, 4th Ed., Transl. by Forbes, p. 167.	Cough and copious muco-purulent expectoration, ever since an attack of pleuro-pneumonia twenty years before.	Dyspnœa. Death sudden, with apoplectic symptoms.	*Left* side of chest one-third smaller than right. Bronchophony around lower angle of scapula.	*Left lung* size of two fists, almost universally adherent by means of fibro-cartilaginous substance. Whole of lung, in appearance and consistence, intermediate between cartilage and fibrous membrane. Two lobes united; the upper of uniform slate grey, and the lower as white as tendon. Bronchial tubes generally dilated, terminating in *culs de sac*—many containing a yellowish opaque matter, and intermixed, a white chalky substance. Mucous membrane of all the tubes dark-coloured and slightly thickened. Smaller bronchial ramifications obliterated. *Right lung* healthy, and very large.
XXIX. M. æt. 57. M. Barth, Mém. de la Soc. de l'Observ. de Paris, t. iii. p. 481.	A toyman; but for last ten years a lamplighter. Originally well-formed and of good colour. Sober and of steady habits. Two attacks of rheumatic fever—one in eighteenth and other in twenty-fifth year. At age of twenty-eight or thirty, spat *two or three ounces of blood;* and since then has had frequent colds, especially in winter. Has become pale and thin—without diarrhœa or sweats. Weakness more marked during last five or six years.	On Jan. 2nd exposed to cold; and seized with shivering, malaise, pain in left side, and copious expectoration (of thick, muco-purulent sputa, frothy on surface). Anorexia, but little fever. Dyspnœa moderate. Pulse 90. On Feb. 14, sharp pain in right side, with increased dyspnœa. Respiration 44. Coughing incessant, and more painful. Expectoration more difficult and less abundant. Gradually becomes worse, with turgescence of face and œdema of extremities. Died on Feb. 20th.	No impulse in precordial region, but clear sound and respiratory murmur. Whole of *left side* of chest contracted. At nipple blue, right semi-circumference forty-three centimetres; and left, thirty-seven. Mobility much less on left. Breath sounds and resonance, generally good in front. Behind (left) dulness on percussion, especially towards base. Inspiration, at times, scarcely audible; at others, loud and sudden. Expiration broncho-cavernous; cough, producing extensive gargouillement, most in subspinous fossa, but diminishing above and below. b *right side,* resonance good posteriorly.	*Left lung* about one-third of natural size, universally adherent by dense tissue, and in front to under-surface of right lung. Of pale red colour, flexible, but tough. In one small point only, a trace of normal lung-tissue. Bronchi generally dilated, but terminating in large spheroidal cavities containing a frothy mucous. Lining membrane smooth, deep red, thick. Intervening tissue slight, much condensed at base. *Right lung* double natural size, seeming to fill almost whole of thorax, and nearly completely covering pericardium. It was congested, œdematous, and friable in posterior lobe. Tissue slightly indurated at summit, and a few tubes have slightly dilated. *Heart* almost square and much increased in size. Valves thickened and opaque. *Ulcerated cancer of lesser curvature of the stomach.*

| cough, especially in winter. | ...soreness, difficulty of swallowing and pain in throat much less, but vomiting worse—only able to take fluid food. Cough and expectoration continued the same, but still not much wasting or alteration of countenance. In last days of Oct. 1848, vomiting became quite uncontrollable, with loss of appetite, rapid wasting, and weakness. Died on Nov. 14. | ...same characters behind sternum and in greater part of left side; but posteriorly, on *left* side, loud mucous rales heard all over, also most evident gargouillement—increasing towards base and completely hiding respiratory murmur and voice-sounds. No dry cavernous respiration heard. | ...many, tongue, touching one another with no intermediate tissue. The membrane lining all the dilatations red, livid, smooth, and thicker than natural. In two cavities, lining membrane partially destroyed, and walls formed by condensed lung-tissue. The pulmonary vessels, within the lung-tissue, seemed to be much contracted. *Right lung* very large, extending to left of sternum and partially covering heart: its tissue and bronchi healthy. *Heart* rather small, and walls of ordinary thickness, though flaccid. Mitral valve slightly thickened. Aorta somewhat dilated, with calcareous plates. *Œsophagus* thickened and contracted at level of sixth vertebra, with surrounding adhesions; much dilated above, with ulceration. Other organs healthy. |

SYMPTOMS.—The symptoms of this affection present a considerable range of variation in different cases, according to the different modes in which the disease originates, and the amount of change which has been induced, not only in the diseased lung, but also in the position and size of the heart. Thus one class of cases—and this includes a considerable proportion of the whole—are chronic from the first, appearing to commence obscurely, and being afterwards characterised by the symptoms of chronic bronchitis, with a limitation of the local signs to one lung. The cough, in these cases, dates sometimes back for a period of twenty years or more.

In another class, the affection dates definitely from some acute chest disease—either a bronchitis, a pneumonia, or a pleurisy without notable effusion—and then goes on, from this starting-point, in much the same chronic way as when the mode of origin is indistinct. The cases in these two classes may or may not be associated with deviation in the position of the heart, signs of enlargement of its right cavities, and dropsy. In a third class, the cases are more acute in their progress, and the morbid change seems to be the immediate sequence of an attack of acute pneumonia. The sufferers included under this last head usually succumb pretty early, and before the disease has attained to its later stages of development. It frequently proves fatal before the end of the first year from the date of the acute pneumonia.

The particular combinations of symptoms in individual cases may be best seen in detail, as they are given in the analytical table. I shall here confine myself to a more general consideration of the different symptoms and signs met with in the disease, and to an estimation of their relative importance.

Although the patient may have many of the physical signs of phthisis, its constitutional symptoms are almost entirely absent. There are no feverish symptoms, no signs of hectic, no copious night-sweats, no disorders of digestion, and the disease for the most part seems altogether of a more stationary and chronic character. No laryngeal symptoms have been noted in any case. Diarrhœa, although an occasional symptom, is less frequent than in ordinary cases of phthisis, and when it exists it may be an accompaniment of blood-poisoning from co-existing gangrene. Once diarrhœa was occasioned by an ulceration of the cæcum, which was rather obscure as to its nature and origin.

Cough is one of the most constant symptoms; it is sometimes present throughout, and undergoes but little variation, though it is often aggravated during the winter months. Where the disease is advanced and there is much dilatation of the bronchi, the cough is often paroxysmal, coming on in violent fits after long intervals of comparative quiet. Such individuals may have violent paroxysms of coughing in the morning, at the end of which the secretion that had accumulated in the dilated bronchi, during the interval between the present and the last fit of coughing, is voided in copious gulps. Vomiting of food may also take place at this time, and one, two, or

even three such fits of coughing may, in some cases, occur during the twenty-four hours. The attacks are preceded by a feeling of discomfort and *malaise*, although comparative relief is experienced as soon as the irritating and pent-up secretions have been got rid of.

Where there is little or no dilatation of the bronchi, the *expectoration* is not very abundant, but rather tenacious, and occasionally somewhat nummulated in character. But where the disease includes dilatation of the bronchi, the expectoration is generally copious, muco-purulent, yellowish, or ash-green in colour; having a tendency to run together into an almost homogeneous mass, which is often frothy on the surface. Owing to the thin sero-purulent nature of the secretion in some cases, the fluid separates, after standing, into three more or less distinct layers—the lowest yellowish, containing most of the solid matter which has settled; a middle stratum of greenish fluid; and an upper frothy stratum, or one composed of mucus and fat granules. In these cases, the amount of fluid excreted daily may reach as much as ten or fifteen ounces. It has often a very stale and nauseous odour, and is sometimes even fœtid,[1] though the smell is quite distinct from that of gangrene. On agitating the recent sputum with water, opaque, greyish filaments, of varying diameter, may soon separate and sink to the bottom. These are casts of minute bronchi, which, as first pointed out by Dr. Arthur Gamgee, assume a purplish tint on the application of iodine. They are met with more particularly in the fœtid sputa, and, according to Niemeyer, the fine acicular crystals of margaric acid may also be detected by the aid of the microscope in the fœtid sputa from dilated bronchi—though they are said not to be encountered in the bronchial secretion in any other lung affection, save that of gangrene. Dr. Grainger Stewart has found these crystals in the dilated cavities after death, but has failed to detect them in the sputa during life.

In more than one-half (17 : 30) of the recorded cases, there has been *hæmoptysis*—sometimes small in quantity, streaking the expectoration, and in others pretty abundant from time to time. Out of the thirty cases there are only four in which there is any mention made of the existence of "tubercle," either in the sound or in the cirrhosed lung.

[1] Upon the presence of what particular substance the fœtor depends, different opinions have been held, as may be seen by the following quotation from Dr. Grainger Stewart's paper:—" Professor Laycock concludes (Edin. Med. Journ., May 1865) from experiments and observations made by the late Professor Gregory, Dr. Arthur Gamgee, and himself, that the odour must be due to butyric acid. He also states that Dr. Gregory detected the odour of methylamine in some of the products of the sputa. Professor Bamberger (Würzburg. Mediz. Leitz. 1864) concludes that the characteristic smell of the sputa in bronchiectasis appears to depend upon a variety of odorous matters, among which are the members of the series of acids of the type to which butyric and formic acids belong, ammonia and sulphuretted hydrogen, all of which may proceed from the decomposition of organic substances. He further states that purulent sputa—*e.g.*, that of tubercular patients—sometimes undergoes the same decomposition out of the body, and, if long kept, have the same smell as the sputa in question. Dr. Arthur Gamgee (Edin. Med. Journ., March 1865), from a considerable number of analyses of sputa, concludes that the occurrence of butyric acid cannot at present be proved to have any semeiological value, and that its presence is in no way characteristic of fœtid bronchitis, under which term he includes bronchiectasis."

In one of these cases (V.) there was no hæmoptysis at all, in another
(XII.) the hæmorrhage seems undoubtedly to have proceeded from the
non-cirrhosed but "tubercular" lung; in the third (XI.) it must, almost
certainly, have proceeded from the cirrhosed lung; whilst in the fourth
(IX.) the hæmorrhage (which was fatal in this case) seems to have
mainly proceeded from the enlarged lung, although there was also
a small cavity containing blood in the retracted lung. Thus there were
fifteen, or one-half of the total number of cases of cirrhosis in which
hæmoptysis was one of the symptoms, and in which the hæmor-
rhage undoubtedly proceeded from the cirrhosed lung, and in only
one of these did "tubercle"[1]—and that in the smallest quantity—
co-exist with the fibroid change. This is an important fact in con-
nexion with the disease, and is in opposition to the view inclined to by
Dr. Walshe and Dr. Law, who have both expressed their opinion as to
the probability of the hæmorrhage, in most cases, proceeding from the
non-cirrhosed lung, in connexion with the formation of "tubercle."
In a small number of cases the patients have complained of pain in
the affected side, either localized or indefinite in site.

Dyspnœa, though a constant symptom, is often moderate in degree,
even in advanced stages of the disease—so long as the patient remains
quiet, and the opposite lung continues to be healthy. It is occa-
sionally more marked as an objective than as a subjective symptom,
and is generally much increased after the slightest exertion. With
reference to the pulse-respiration ratio, no definite details are given,
except as to its condition in the case recorded by Dr. Walshe. Here
he says, "It never fell lower than 3 : 1, and was sometimes found at
the par of health, 4 : 1; even above this on one occasion—47 : 1."
The dyspnœa is most marked in cases where there is dilatation of the
right side of the heart and dropsy; orthopnœa is then a constant
symptom, attended with more or less lividity of lips, face, and even
surface of the body generally, whilst there may also be pulsation in
both jugular veins. Purpuric spots of hæmorrhagic effusion appear
on the body occasionally. When an acute attack of bronchitis or
pneumonia supervenes, the dyspnœa becomes asphyxial in its inten-
sity, owing to interference with the breathing power of the previously
sound lung, and death often speedily ensues.

The patient almost habitually lies on the retracted side; and any
attempts to lie on the other cause great increase of dyspnœa and
cough, so as to make it impossible to continue in this position.

The pulse is often regular and full, notwithstanding the frequent
deviation in position of the heart. The appetite is usually pretty
good; and in spite of the chronic nature of the cough, and the almost
habitual copious expectoration, the patient does not lose much flesh.
Towards the end slight emaciation is common, but extreme emaciation
is rare in this disease; when it is uncomplicated by cancerous or
other wasting affections.[2]

[1] Really, in all probability, a patch of chronic lobular pneumonia.
[2] Since this was written I have seen and made the autopsy of a man who suffered from

The mode in which the third class of cases originates has been well described by MM. Grisolle and Charcot. The individuals do not recover from the attack of acute pneumonia as they do in ordinary cases. On this subject the former observer says : " One sees at first the disease decline—in appearance at least ; the pain in the chest disappears ; the sputa lose their viscosity as well as their hæmorrhagic colour ; the appetite reappears ; but notwithstanding this improvement some symptoms obstinately persist ; the patient, far from gaining flesh and strength, grows worse and worse in these respects, and one finds, on examination of the chest, that a more or less considerable portion of the lung still remains impermeable to air—that is to say, percussion reveals dulness for a certain extent, whilst over the same part, on auscultation, bronchial respiration and bronchophony, with sub-crepitant and mucous râles, are heard." But it must be clearly understood that it is not the mere persistence of the local symptoms alone which have any significance, since M. Grisolle has shown that a slow return of the lung to its normal condition is a common, if not an habitual, sequence in a pneumonia whose result is favourable. Feebleness of the vesicular murmur, and a coarse breath-sound, mixed with sub-crepitant râles, are often the only signs of the unfinished resolution ; though much more rarely, as M. Charcot says, " tubular breathing, bronchophony, and a more or less marked dulness, have been capable of persisting for two or three months after the complete cure of a pneumonia, and, notwithstanding this, there has not been the least tendency to a relapse, or the least return of febrile symptoms." Cases of this kind, however, which are not those to which we are more especially alluding, may be interpreted, as M. Charcot believes, by supposing that the new consolidating " materials have not been re-absorbed, and have remained for a time in the tissue of the lung, without the co-existence of any inflammatory action." But, in the cases where an inflammation of the lung is about to terminate in what MM. Grisolle and Charcot term "chronic pneumonia," (or, as we prefer to say, in a fibroid induration leading to cirrhosis), although the general symptoms occasionally subside for a brief period, they soon reappear. The symptoms, then, have more or less of a hectic character from the first ; or there may be a preliminary and short reappearance of the symptoms of the acute condition —in other words, a relapse, of short duration. Gradually the hectic symptoms become more marked : every evening the skin becomes hot and the face flushed ; sometimes night-sweats are profuse, and at others they are absent altogether ; nutrition soon becomes impaired and the patients lose flesh, whilst cough and dyspnœa continue. Œdema of the lower extremities may supervene, and the patient, already wasting, may be still further lowered by the setting in of an obstinate diarrhœa. The resemblance of the general symptoms to those of

in extreme degree of cirrhosis of the left lung, and in whom there also existed an enormous liver, studded throughout with the most typical cancerous nodules. No cancer was found in any other organ except in the bronchial glands, which were completely infiltrated—not even a trace of it could be discovered in either lung.

PHYSICAL SIGNS.—*Retraction or shrinking of t*
or less extent, on the side of the affected lung,
the disease has existed for a certain time. It
monly met with till after the lapse of about eigh
only out of seven cases, which proved fatal at
was there any flattening of the chest. One of tl
child only seven years old, at which age of cours
would readily follow the shrinking lung; whi
(IV.), that of an adult, although the disease onl;
for nine months, it seems to have made rapid p
obvious shrinking of the chest on the affected
lowering of the shoulder. In the great majori
who live longer than eighteen months after the
disease, some amount of retraction of the che
general or sub-clavicular; and in almost all, th
amount of immobility on the retracted side. M
where contraction cannot be detected, comparati
easily established. The flattening and retractio
physical process dependent upon the shrinking o
its amount depends principally upon the degr
thoracic parietes at the onset of the malady, and t
course. If the lung-shrinking goes on pretty ra
is young, the amount of contraction may be enor
curred in Dr. Mayne's case (XXIII.), where the
six years, and had commenced when the patie
years old. The more the neighbouring viscera ar
gradually vacated by the shrinking lung—the n
enlarges—and the more the actual amount of
minished by the formation of dilated bronchial
be the amount of contraction or flattening of the
these conditions must be considered together,

lung, which extends into the diseased side of the thorax. In some extreme cases the percussion note may be good over almost the whole of the affected side in front, whilst it is absolutely dull with characters of resistance posteriorly.[1] On the opposite side of the chest the percussion note is almost always clearer than usual, and more like that which is met with when the subjacent lung is emphysematous.

On *auscultation* the normal respiratory murmur is either altogether absent or heard only over limited areas; whilst at other parts the respiration is high-pitched and bronchial, with cavernous and amphoric characters here and there. These sounds may be of the dry character; but, more frequently, there are moist rhonchi of various kinds—sometimes smallish, but mostly of the large bubbling kind and of a metallic character—such as constitute what is frequently described as *garpouillement.* The loud bubbling râles may be so abundant as to drown almost every other sound. Vocal resonance may be either diffused and bronchophonic, or various degrees of pectoriloquy may exist. Vocal fremitus is generally much increased over the dull parts, and this, together with the great sense of resistance on percussion, has, when it exists to a marked extent, considerable diagnostic value. The signs indicative of cavities may exist most plainly under the clavicle, towards the middle of the lung, at the base—or, perhaps, in all of these situations at the same time. It can scarcely be said, positively, that they are more frequent in one situation than in the other. Sometimes such signs, however, may be absent altogether. The breath-sounds over the opposite enlarged lung mostly deviate from the condition of health, only by being louder and more puerile than natural. When an intercurrent attack of bronchitis or pneumonia sets in, the character of the respiration on this side will, of course, undergo a corresponding modification.

The *position of the heart* often deviates much from that which is normal. The amount of displacement, of course, depends in great part upon the amount of lung-shrinking; but it is generally more considerable when the disease is in the right lung than when it is in the left. When the right lung is affected, the whole heart seems to be drawn over bodily into the right side of the thorax, so that its impulse may be felt only to the right of the sternum, whilst its apex impinges under the nipple. An amount of displacement so considerable as this has been encountered several times. In one case in which the disease was on the left side, the heart's impulse was perceived close under the left clavicle; but in other instances the organ seems only to have been slightly drawn up, and the area of impulse, therefore, only slightly raised. Though its position is altered, the heart mostly beats with regularity, and no bruits seem to be produced by the displacement.[2]

[1] This was most notably so in two cases (XXIX., XXX.) recorded by M. Barth.
[2] Dr. Andrew Clark, however, states that "a low-pitched systolic bruit is commonly heard over the pulmonary artery."

More or less dilatation and hypertrophy of the right side of the heart has occurred in one-third of the cases. The deviation in position of the heart would often make it difficult to establish this by percussion and auscultation, but in three of the cases (VI., XIII., XXII.) there were the signs of a loud tricuspid regurgitant murmur, associated with pulsation in the jugular veins and more or less dropsy. And in almost all the cases where the right side of the heart was found to be enlarged after death, there had been dropsy during life—either anasarca alone, or anasarca and ascites combined in a few of the cases. Dropsy existed in more than one-third (12 : 30) of the cases; so that it was present (mostly in the form of anasarca of the lower extremities) in a few cases where there was no dilatation of the right heart, and in which it depended upon disease of the kidneys or other co-existing conditions.

In the *acute form* of the disease, answering to what has been called " chronic pneumonia " (which may affect the whole of one lung, or only one lobe[1]), the physical signs are almost identical with those of the early stage of the chronic form, before much contraction of the lung has taken place. Thus, there is absolute dulness over the diseased part, with a considerable sense of resistance to the finger, whilst the vocal fremitus is much intensified. On auscultation over the dull part, bronchial or tubular breathing is heard; the latter being sometimes so loud as to be of a cavernous character even where no cavities exist. Râles are generally heard also—often loud, large, and metallic, though they are sometimes smaller or even absent. The vocal resonance is mostly bronchophonic. Occasionally, however, there may be a *complete absence of all breath-sounds*, either healthy or morbid, and of vocal resonance, whilst the percussion sound is quite dull—a combination which occurred in a case observed by M. Requin and quoted by Grisolle, and which led to its being mistaken for one of pleuritic effusion. In another case (III.), which has been recorded by M. Charcot, remarkable alterations were observed on different days. At one time, over the whole extent of a lung which was diseased throughout, there was a complete silence—no sound or râle of any kind; whilst at other times, on the contrary, there was loud and universal tubular breathing mixed with metallic râles. Unfortunately no observations were made as to the state of the expectoration at these times—either as to its quantity or quality.[2] Contraction of the chest walls is of course not met with until the diseased lung has undergone a certain amount of shrinking, and by that time, if the patient survives so long, the intensity of the general symptoms has

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[1] In this latter case the lower lobe of the right lung is said to be the most frequent seat of the disease.

[2] Charcot says:—"Cela eut été cependant fort intéressant ; car dans la pneumonie aigue, où l'absence de tout bruit respiratoire, normal ou anormal, s'observe quelquefois, ce phénomène paraît en général dépendre de l'obstruction des tuyaux bronchiques des parties hépatisées par une grande quantité de liquide visqueux ou par un bouchon d'exudation concrète." A similar temporary silence has been occasionally observed in cases of bronchiectasis.

inished, and the condition comes to resemble that of a person
o is suffering from the more chronic form of the disease.

DIAGNOSIS.—The diagnosis of this affection in certain well-marked
es, can be made almost with complete certainty, though in other
ances only with great difficulty. The diseases with which it is
st likely to be confounded are chronic pleurisy with retraction of
 side, cancerous infiltration of one lung, certain forms of "tuber-
ar" phthisis, simple general collapse of one lung, and simple or
nary bronchiectasis.
n chronic pleurisy with retraction of the side, according to Dr.
lshe, the ribs are twisted downwards and inwards, the spine is
ved and the shoulder is drawn down; which effects are not pro-
ed by Cirrhosis alone. The lowering of the shoulder was, however,
tinctly produced in one case of Cirrhosis occurring in a youth, with
om the amount of chest contraction was extreme; and it also ex-
d to a slight extent in another case. Cirrhosis being so frequently
nplicated with dilatation of bronchi, is more frequently associated
h physical signs of the hollow class: though the bronchial symp-
ns are not always most severe in cases where there is the greatest
ount of contraction of the chest walls. The heart is generally
ich more displaced by Cirrhosis than by chronic pleurisy. Then
e frequency of hæmoptysis in Cirrhosis, with its non-occurrence in
ronic pleurisy, must be borne in mind; and also, the greater fre-
ency of enlargement of the right side of the heart with dropsy in
e former affection. As Dr. Peacock has suggested, it will be well
so to bear in mind the possibility of confounding Cirrhosis of the
ing with contraction of the organ succeeding an empyema which is
acuating itself through the bronchi, by means of a fistulous com-
unication between them and the pleura.
Cancerous infiltration of one lung also causes retraction of the
de, though, according to Dr. Walshe, the retracted ribs are not
tered in axis, and the tendency seems to be to draw in an upward
rection rather than laterally, so that when the disease occurs in the
ght side, the liver may be much elevated, though the displacement of
e heart is much less than is met with in Cirrhosis. Occasionally,
wever, as we have seen, the same upward traction occurs in Cir-
osis. In both there is frequently cough, expectoration, failure of
trition, and often hæmoptysis. The existence of well-marked signs
cavities of a stationary character, such as are due to dilated bronchi
 Cirrhosis, would be absent almost universally in cancer. The
ndition of cachexia is generally more marked, however, in cancer,
 well as the amount of intrathoracic pain; and the disease is often
ore rapid in its progress, its duration being sometimes much less,
d never exceeding two and a half years. As Dr. Walshe points out,
o, cancer of the lung is generally associated with a mediastinal tu-
ur of the same nature, so that not only may the morbid percussion
te extend across the middle line, but there is apt to be greater

dyspnœa, with lividity of face, and other pressure signs—such as
dilatation of the superficial veins, and œdema of the thoracic parietes.
In cases of cancer of the lung, moreover, cancerous tumours may
exist in other parts of the body, and towards the last the cancerous
cachexia often becomes extreme. This affection could, therefore, as
a rule, only be confounded with the more acute forms of Cirrhosis.

"Tubercular" disease of the lung, presenting such characters as
would render it liable to be confounded with Cirrhosis, is only
encountered with extreme rarity. The characters of the latter disease
which are most opposed to those of the more ordinary forms of
phthisis are the signs indicative of an almost absolute freedom from
morbid deposit in one lung, combined with the gravest amount of
implication of the other—producing, perhaps, not only retraction of
the side, but also cavities, and more or less complete impermeability
of the lung-tissue between them. Then with local signs of so pro-
nounced a character on one side (whilst the other lung appears to
remain intact), we not only have no laryngeal disease, but there is a
comparative absence of the constitutional symptoms peculiar to
phthisis: so that there is an utter disproportion between the gravity
of the local and the constitutional signs, and at the same time the
disease presents a comparatively stationary character. Cirrhosis also
frequently exists in previously strong individuals with well-formed
chests ; and, in one-third of the cases, there have been signs of
hypertrophy and dilatation of the right heart, associated with dropsy.
Only the contraction of an enormous tubercular cavern could produce
such an amount of displacement of the heart as we frequently meet
with in Cirrhosis; and that such an amount of disease and disorgan-
zation of one lung as this implies, should have existed without the
least implication of the other, is contradictory to all experience as to
the nature of ordinary phthisical affections. Any great contrac-
tion occurring in a "tubercular" lung is almost certain to be due
to a considerable admixture of fibroid substitution with the other
morbid product, so that the points of diagnosis just considered may
be said to be those distinguishing the pure fibroid from the mixed
fibroid and "tubercular"—or rather fibroid and pneumonic forms
of phthisis.[1]

Simple general collapse of one lung is a condition of extraordinary
rarity, which, as Dr. Walshe says, could only result from the pressure
of an aneurism or a tumour upon the main bronchus. In such a case,
in addition to the signs of the tumour which might exist, there would,
in all probability, be a dull, toneless sound on percussion, instead of
resonance of a wooden or even tubular character, whilst the respiration

[1] With reference to the presence or absence of diarrhœa, it will be well to bear in mind
the following remarks by Dr. Andrew Clark, which are in perfect accordance with my own
experience.   Dr. Clark says (Trans. of Clinic Soc. vol. i. p. 188) : "Experience has
peremptorily taught the writer, that the occurrence of ulceration of the bowels in the
course of chronic disease of the lungs is not conclusive as to its tubercular nature. De-
posits in and ulcerations of the intestinal glands may occur in almost any form of chronic
disease to which the lung is liable."

would be simply weak, instead of bronchial, with more or less signs of cavities.

Simple primary bronchiectasis of one lung may exist, and then be followed by more or less fibroid induration of tissue.[1] Many of the signs and symptoms of this disease would be similar to those of Cirrhosis; only, in the early stages, the signs of cavities would be marked, whilst those indicating consolidation of the intervening lung-tissue would be comparatively slight. The signs of retraction of the chest and displacement of heart are almost or completely wanting.

PROGNOSIS.—In almost all cases, the individuals suffering from this disease are ultimately carried off by an acute affection of the hitherto sound lung. An attack of bronchitis or a pneumonia supervenes, or a mixture of these two conditions, and the breathing power becomes so seriously interfered with, that the patient rapidly dies in an asphyxiated condition. Death may take place, also, from gangrene in the cirrhosed lung; or a copious effusion of blood proceeding from an ulcerating cavern in the lung may prove fatal—although usually the amount of blood lost in this way is not extreme.

In those cases hitherto styled " chronic pneumonia," and in which an extreme amount of fibroid induration follows an attack of acute pneumonia, the patient is apt to die in a state of marasmus, or from uncontrollable diarrhœa, before the local disease has attained its maximum—that is to say, before much contraction of the lung has occurred, or many bronchial caverns have been formed.

Death may also take place, however, when the disease is fully established, without the advent of acute inflammation in the opposite lung, but gradually, owing to the mere exhausting influence of the disease—when it is associated with marked bronchiectasis, and when the amount of purulent fluid daily expectorated is extreme. The occurrence of dilatation of the right side of the heart to such an extent as to produce tricuspid regurgitation, is, of course, a most grave complication.

In other cases, the patient is cut off by some acute or chronic co-existing malady, such as disease of the brain, cancer of the stomach, or uncontrollable diarrhœa from ulceration of the cæcum—diseases which actually proved fatal in a few of the cases included in my list.

Although the ultimate prognosis in this disease is most grave, still, if the sound lung can be maintained in its condition of health, the fatal termination may be warded off for some time, and the individual may live for years after the disease has been fully established.

TREATMENT.—The indications in this disease are to pay prompt attention to the very earliest signs of bronchitis, or pneumonia in the

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[1] In rare instances, as before stated, owing to the amount of secondary induration and contraction, some of these may actually develop into cases where the cirrhosis becomes the most prominent feature.

non-cirrhosed lung, so as, if possible, at once to arrest its progress. The patient's life should, moreover, be so regulated that, whilst exposed to wet and cold as little as possible, he may be brought under the influence of habits which are best calculated to promote the general health. The development of the non-cirrhosed lung should be favoured by such carefully regulated exercise as can be indulged in without distressing the heart's action, or causing much dyspnœa. Plenty of time should be spent in the open air; the diet should be good, simple, and nourishing; and the functions of the skin should be stimulated by the daily use of baths and dry friction. Whilst these general measures are being adopted, their action may be supplemented, when necessary, by various medicines. The dilute mineral acids or salts of iron, combined with bitter infusions, or iron and quinine, may be had recourse to; whilst in some cases, cod-liver oil, either alone or combined with iron, will be of much use.

In cases where diarrhœa sets in, every effort must be made to arrest this by the careful administration of dilute sulphuric acid, or by opiates and the various vegetable astringents; and, in like manner, where dilatation of the bronchi is well marked, and the daily flux from these is excessive, we must endeavour to check the copious flow by the administration of astringents combined with balsamic remedies (such as tolu, copaiba, or turpentine), and an application of counter-irritants externally. Where necessary, also, we must endeavour to bring about a regular and periodical evacuation of the dilated bronchi; so as to prevent decomposition of the retained secretion within the tubes, which is liable to produce general distress, and may also entail local gangrene. For this purpose Niemeyer strongly recommends the inhalation of turpentine two or three times a day. About half a drachm of the spirits of turpentine is to be placed in a bottle of hot water, and by means of some suitable addition to the neck of the bottle its vapour is to be inhaled. In this way the amount of secretion not only is diminished, but violent fits of coughing are induced in from ten to fifteen minutes, which are accompanied by an evacuation of the contents of the dilated bronchi. Niemeyer says he has seen great amelioration thus induced in the symptoms of patients whose condition had been previously most distressing.

With regard to the possibility of bringing about an actual disappearance of the new fibre-tissue, and a reappearance of the lung-tissue which it has supplanted, this seems a result beyond our most sanguine expectations, and one to which we are scarcely likely to attain. But, whilst the disease is still advancing, we may hope and ought to endeavour to prevent the spread of the morbid change to previously healthy portions of lung-tissue. This desirable result will be best brought about, not only by the means before alluded to, which are destined to bring the patient's general health up to the highest possible standard; but will, perhaps, be also encouraged by the use of iodide of potassium internally, in conjunction with counter-irritation to the affected side, and the free inunction of iodine locally. As Dr. Walshe

ests, a trial might also be made of some of the natural iodurated
ers, such as those of Kreuznach or Woodhall The amount of in-
ace which the iodides have in checking the over-growth of fibre-
ie seems in some cases to be most marked, and in a disease of so
e a character as this we are bound to try the influence of reme-
 which may have a favourable action, so long as they exercise no
terious effect.

APNEUMATOSIS.

·By GRAILY HEWITT, M.D., F.R.

DEFINITION.—APNEUMATOSIS is that conditior
characterised by the return of certain air-cells tc
the portions of lung so affected have once been ɪ
and efficient in promoting ther espiratory chang
lating through them, and have ceased to be so.

HISTORY.—The older observers of the disease
the great frequency with which they found after
of the lungs solidified. The death was in sucɦ
this alteration of the lungs; and as it resembled,
ward appearances observed, the solidification foɪ
adults, and which had received the name of
naturally enough gave the two conditions the
within a comparatively recent period has it be
the two conditions are essentially different.

One circumstance, however, was observed as
solidation was always in the cases of young ɑ
abruptly separated from the adjoining sound lun
as it were by the lobular divisions of the lungs.
"lobular pneumonia." It was said that in the
young children the pneumonia was *lobular*. Thɩ
disease so-called was found always to be very con
children were considered to be *par excellence* predis]

Seifert [1] recognised the nature of the so-called lobular pneumonia so far as its mode of production was concerned, giving it the name of "bronchio-Pneumonie," and he pointed out the counterpart of the lesion in that kind of pneumonia seen in adults which Laennec termed "peripneumonie des agonisants," and Piorry "pneumonie hypo-statique." There is reason to believe, however, that some of the cases alluded to by him were really cases in which the lung had never been expanded at all at the affected parts, but remained from the day of birth in the state described, and truly explained by Jörg as *atelectasis.*

Barthez and Rilliet [2] first, in 1838, distinguished between "lobular" and "lobar" pneumonia, laying down the principle that lobular pneumonia was always a secondary affection connected with bron-chitis.

MM. Legendre and Bailly,[3] however, have the merit of first pointing out the essential nature of the condition known as lobular pneu-monia. They used the very simple expedient of artificially inflating the lungs after death, and observed the effect of the inflation on the portions consolidated and in a state of lobular pneumonia. The result was, that the apparently hepatized parts swelled out, became filled with air, and were, as it seemed, suddenly converted into healthy-looking lung-tissue. The lung so inflated was found to possess all the physical characters of lung in a normal condition, and it was evident to these observers that the essential difference between the pseudo-hepatized and the sound lung only consisted in this,—that in the former case the air was withdrawn from the air-cells, the tissue of the lung itself not being necessarily altered. They were led to this result by observing how closely the lobules so collapsed resembled in external characters those of the lungs of the foetus which has never respired; hence they replaced by the term "état foetal" the old designation "lobular pneumonia." The results of this discovery have been most important in enabling us by a simple and easily applied test to ascertain whether the condition of the lung present in a particular case is one only of collapse, or whether it is due to a change of another character altogether.

MM. Legendre and Bailly showed that in the cases in which their "état foetal" was present, there was no true inflammation of the lungs present such as would entitle them to be considered as cases of true pneumonia; pneumonia, as it is generally understood, being con-stituted by a breaking down or softening of the lung substance, whereas in cases of lobular pneumonia the lung-tissue is quite firm, and, with certain exceptions, not easily broken down under the finger. The peculiar limitation of the morbid change to certain lobules, the change beginning and ending abruptly, and not shading off gradually, also, evidently gave to it a character altogether distinctive, and such

[1] Die Bronchio-Pneumonie der Neugeborenen ; Berlin, 1837.
[2] Traité des Maladies des Enfants.
[3] Archives de Médecine, 1844, p. 157.

fœtal." The term "Apneumatosis" was first e
an excellent treatise on the subject, and its adop
by myself in a paper read before the Royal M
Society of London.[2] The old term "lobular p
culated to give very erroneous ideas of the co
applied, a new term was necessary. "Pulmor
sufficiently distinctive, the word "collapse" bei
very properly, in another sense, to indicate the
of the lungs, which always occurs to a gre
opening the thorax after death. In the abs
word Apneumatosis (*a, privative*, and πνευματω
filled with air) was considered the most approp
does, precisely the condition which is present,
theory as to its cause or mode of origin.

Apneumatosis is not simply an anatomical a
regarded as a diseased condition of the lung n
variety of symptoms and signs, producing certai
the system at large, and very frequently provi
ance fully justifies its being considered separ
with which it is always, or almost always, assoc
of early childhood is, when fatal, almost inv
Apneumatosis.

PATHOLOGICAL ANATOMY.—A description of
of those portions of the lungs affected with
reality include more or less completely a descri
variously designated by authorities up to the p
inated *Lobular Pneumonia, Carnification, Pul*
fœtal," Marginal Pneumonia, Catarrhal Pne
Bronchio-Pneumonie (Seifert), together with som
Atelectasis, all these terms applying to one col

In the first place, the lobules affected are remarkably distinct; abruptly separated from adjoining healthy lobules, generally depressed below the surface of the healthy lobules; for the most part they are found at the margins of the lobes,—those portions of the lobes most distant from the root of the bronchial tree. The distribution of the affected lobules is evidently connected with their relation to the divisions of the bronchial tubes, and is such as to preclude the idea of the affection having spread by simple contiguity. The lobules supplied by one particular bronchial tube all present the physical characters of the lesion, whilst the lobules supplied by the closely contiguous bronchial tube may be perfectly healthy. The number of lobes affected is of course subject to great variety: it occasionally happens that the whole of one particular lobe is affected, but this is not very frequently observed, and it most commonly happens that nearly all the lobes present a greater or less number of apneumatic patches, although it is somewhat rare to find apneumatic portions present in all the lobes.[1] Certain parts of the lungs present this lesion with remarkable frequency. These are, first, the lower margins of the lower lobes of both lungs, the tongue-like prolongation of the upper left lobe, and the middle lobe of the right lung. Next in order come the posterior surfaces of the lower and of the upper lobes. The order of occurrence here laid down is rarely departed from, a very interesting circumstance, and one which will again be alluded to in considering the essential nature and mode of production of the lesion in question. The lobules situated at the periphery of the lung are thus the first affected, and in slight cases the alteration is confined to them.

In many cases the apneumatic patches are symmetrically placed on the corresponding parts of each lung. This is especially the case with the posterior surfaces of the lower lobes, where we have frequently observed a line concave superiorly passing across this aspect of the lobe on each side, and forming the upper boundary of an apneumatic portion of the lung almost identical in form and equal in superficies on the two sides.

It generally happens, when Apneumatosis of the kind to which the description given above would apply is present, that the healthier parts of the lungs are pitted and depressed at certain parts, and these depressed portions have a rather darker colour than usual. The little pits are caused by the partial collapse of the air-cells there situate, and, although slight in degree in particular lobules, the aggregate number of air-cells thus rendered useless may be very considerable. Apneumatosis thus slight in degree disappears completely on inflation, and between this and the more complete form involving the whole of several adjacent lobules, many gradations may be witnessed.

To the naked eye the apneumatic patches appear like islets of a darkish red colour abruptly separated from the lighter and more healthy lobules, and having a sharp determinate outline. On a more minute

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[1] See Valleix, op. cit., p. 62 et seq.

sent often depends on the degree to which the lobule
Sometimes the colour is a lightish red, like that of a
muscle, but at other times it is a deep purple ; and be
extremes many varieties of colour are observed.

The depth to which the lung-tissue is affected is in
ratio to the degree in which the lung is seen to be ap
ficially. Section of the part shows the same definite
Apneumatosis which is evident externally ; the shape
lung involved in the change is determined by the outli
to which certain bronchi are distributed, and each apn
has thus a more or less pyramidal form, the base of th
towards the periphery. The apneumatic lobules are ge
below the level of the adjacent part of the lobe. Th
the case, however. When these lobules are less in
they have a somewhat lighter colour than in the ot
things. Thus the apneumatic portions which are
colour have usually the normal bulk, and may even
circumstance is connected with the greater or less qu
contained in the vessels of the part.

The *consistence* of the apneumatic lobules is open to
This is in like manner connected with the quantity
the vessels of the part, and also with the length of
has existed. The paler, less bulky lobules have a loose
resembling that of a piece of flesh : the anterior to
upper lobe often presents this peculiarity. The mor
darker parts have, on the contrary, a very firm co
resembling that of a piece of liver, and resist press
effectually than the looser portions. In all cases
lobules are found to sink in water. The section is
even, and when the part is much congested it resembl
clot of blood. The bronchi cut through often contain
considerable quantity. Where the lobe is thin, as
tongue of the upper lobe, the lobules are in many cas
tinctly between the fingers. This is due to the fact th

of the air-channels is chiefly found in those parts in which less congestion is evident.

Inflatability.—The most important circumstance, however, in reference to the apneumatic lobules is the absence of air. No air-cells are visible on the pleural surface, none on the surface of the section. The difference presented between two parts of the same lobe, one of which is apneumatic and the other healthy, is in this respect most remarkable. All around the affected lobule the air-cells are most distinct and evident: none are visible where the Apneumatosis exists. These lobules are, in fact, as a rule completely destitute of air; the opposite walls of the air-cells are in apposition; the finer air-tubes are either filled with fluid or in the same condition as the air-cells themselves—*i.e.* collapsed. As a consequence of this non-aërated state of the lobules, they are found to be absolutely non-crepitant on pressure. If a blow-pipe be introduced into the bronchus leading to the collapsed portion, and air be then forcibly injected, an instantaneous change takes place in the colour, appearance, and physical characters of the apneumatic lobules: they swell out, become of a light rose-red, air-cells are at once visible on the surface, and the affected lobules come to so far resemble the adjoining healthy ones, that they are not to be distinguished from them. As has been already stated, this effect of insufflation was first pointed out by MM. Legendre and Bailly. Now the effect of this insufflation is not always the same. It is not always entirely successful, some portions of the affected lobules resisting this forcible refilling of the air-cells. As a rule, the operation is performed easily and with the use of very little force, and it always partially succeeds. The portions which resist the attempt to introduce air are those which are the most congested, and in these small portions remain uninflated whatever may be the force used by the lungs of the operator. This point it is important to remark upon, for there is reason to believe that a want of success in the operation of inflation in cases of the kind alluded to has induced some observers to doubt the correctness of the general statement with reference to the nature of the lesion now under discussion. The fact is, however, that where the Apneumatosis and considerable congestion co-exist, the pressure of the contents of the blood-vessels effectually prevents the re-distension of the air-tubes, and air cannot be made to pass into the ultimate air-cells. A difficulty of the same kind occurs when the air-tubes are much distended with fluid secretion, but here it is hardly ever practically productive of much opposition to the operation of inflation. After inflation has been performed the dilated air-cells have a tendency to collapse again in a short time if the bronchus leading to them be not tied.

The operation of inflation is to a certain point a test of the presence of Apneumatosis as distinguished from pneumonia. In Apneumatosis, as has been already explained, it generally succeeds. In pneumonia, however, the lung-tissue cannot be restored to its *natural appearance* by inflation. It is not often that an opportunity presents itself of

as rare to find it free from disease.

The physical characters of Atelectasis as dist
Apneumatosis.—The two conditions are almc
cally speaking, and in some instances, indeed,
particulars of the case must be examined in ord
at issue. Some go so far as to say that Apneu
are one and the same thing, that the lobules pr
described above as those of Apneumatosis have
at all, and have been in the condition in which
grounds on which this statement is made are
will not bear examination. The following fact
settle the contested point : a child, previously
a catarrhal affection of the air-tubes ; up to th
evidence that the act of respiration has been h
a regular manner; after a few days the child d
of all the lobes is found collapsed ; the physica
during the last few days, having indicated, s
gressive and increasing disease of the lungs. A
in every particular, it is not a difficult matter
an amount of lung implicated in the lesion it
that symptoms could have been previously ab
affected with atelectasis is easily the prey t
latter affection is so fatal because in such a case
by Apneumatosis. This seems to be the prop
relation of the two lesions, atelectasis and
the other.

When the lung has undergone mechanical
become hardened, reduced in bulk, as in cases of
its physical characters to a certain degree rese
matosis. It seems desirable to restrict to t
carnification. Carnified lung is firmer and de

resembling at first sight tubercular masses. These little cavities are situated immediately under the pleura, communicating freely with the bronchial tubes. They are the "granulations purulentes" of Fauvel. They are for the most part, according to our own experience, found in portions of lung affected with Apneumatosis, but they may be found in other situations, and are not therefore perhaps so entirely a part of this affection as to justify their being considered fully in this place. It will suffice here to say that in chronic cases in which Apneumatosis is present the little cavities in question are rarely absent. They are often described as "bronchial abscesses," "vesicular bronchitis," &c.

ETIOLOGY.—Apneumatosis is a mechanical effort of the presence of certain morbid conditions of the air-tubes, these morbid conditions appearing to be particularly efficacious in the production of Apneumatosis during infancy and early childhood.

Catarrhal inflammation of the bronchi, either existing *per se*, or forming a part of other diseases, is a very common affection in early childhood, and Apneumatosis is one of its effects, the presence of a mucous secretion in the finer air-tubes preventing the due aëration of the lobules to which they lead. The connexion between the two circumstances, excessive secretion and collapse of the air-cells, is one which is supported by considerations, the result of experimental and pathological inquiry.

It appears that any obstruction of the bronchial tubes is sufficient to produce after a time the appearances of Apneumatosis in the distal lobules. The experiments of Mendelssohn and Traube, described by Fuchs,[1] are especially interesting as demonstrating this fact.

In one of these experiments tracheotomy was performed on a dog, and a shot introduced which was afterwards found in the left bronchus. In two days death took place, and the appearances found were as follows :—The right lung was emphysematous, enlarged ; the left lung was collapsed, its lower lobe was in great part congested, devoid of air, and also the upper lobe in certain parts, near which lay emphysematous patches. Inflation distended the whole lung. In other experiments, a like effort was produced by the introduction of a ball of paper, certain portions of the lungs becoming hard, condensed, and no air-cells being visible on the surface.

The resemblance between these cases and those of children affected with Apneumatosis due to the obstruction produced by the bronchial secretion is, as Fuchs remarks, at once apparent. The relation of bronchial obstruction to pulmonary cohesion, also indicated by Legendre and Bailly, has been more completely developed by Dr. Gairdner,[2] so far as the mechanism of the process is concerned, with whose acute and original remarks our own almost completely agree. Dr. Gairdner has demonstrated the nature of certain lesions of the

[1] Loc. cit. p. 61 et seq.
[2] On the Pathological State of the Lung connected with Bronchitis and Bronchial Obstruction : Edin. Monthly Journal, 1850-51.

stress on the imperfect inspiratory power of
additional predisposing element in the produc
question. Gairdner's satisfactory and lucid
rationale of the process by which Apneumatos
lung, is produced, is as follows.

Commenting on the experiments of Mendelsso
alluded to, he says, "It is clear, therefore, from
from pathological observation, that the most
effect of obstruction, or of diminished calibre of
caused, is not accumulation" (as Laennec l
diminution in quantity of the air beyond the ol
author then shows that another mechanical d
into play in producing collapse from obstruct
the form of the tubes; these diminishing in
the periphery of the lung; consequently, if the
nearly filled at one point by a plug of mucus
tion, propelling it towards the air-cells, will l
the tube when it arrives at a part, the calibre
that which it originally occupied. The plug of
a ball-valve, and at every expiration a portion
which, in inspiration, is not replaced. In tl
which the bronchus in question leads, contains
condition to which it is reduced is one of Apr
the work referred to, and also quoted by Gai
disappearance of the air from the lobules, l
absorbed by the blood-vessels, having been firs
by the presence of mucus in the tubes, these l
their calibre diminished by the thickening of
always present. Dr. Gairdner's explanation
dependent on the supposition that the broncl
viscid material: this may be the case in adu

theories of both of these authors. If such absorption take place, it is natural to suppose that the oxygen will disappear first, and be replaced by carbonic acid: this later product being readily dissolved in fluid will also finally be carried away, together with the nitrogen. The dark colouring of the apneumatic portions Fuchs attributes to the excessive quantity of carbonic acid present.

The inability to cough and expectorate is another circumstance to which Dr. Gairdner alludes as a cause of bronchitic collapse. It appears to us, however, that this is rather to be looked upon as a consequence than as a cause of the collapse, at least at the commencement. The efficiency of the cough in expelling mucus from the tubes is dependent on the presence of air in that part of the tubes beyond the obstruction. Each lobule is a miniature lung, and the sudden expulsion of the air from the lobules drives the obstructing agent before it. As long, therefore, as the air-cells contain air, so long will the cough aid in the expulsion of mucus from that part of the lung. When the Apneumatosis has been produced in certain lobules, those lobules are in great part unaffected by the cough, and there is no expulsion of mucus from the air-tubes with which they are supplied. The Apneumatosis is thus *perpetuated* by the inability to cough and expectorate, but it is not produced by it except in a secondary manner. The fatal result of cases in which Apneumatosis occurs is probably connected with the absence of expectoration, and the imperfect character of the cough.[1]

It is evident that the condition here supposed to be effective in the production of Apneumatosis is only the last step of the process. Why, it will be inquired, is Apneumatosis so especially common in young children, while it is so rarely observed in adults. In the first place, it must be answered that Apneumatosis is not so rare in adults as has been imagined, which fact is shown by a perusal of Dr. Gairdner's papers just alluded to; his statement being in great part, indeed, founded on observations made in adults. But, on the other hand, it cannot be denied that Apneumatosis is comparatively much more common in early life, and there must accordingly be certain powerful predisposing circumstances leading to this result, favouring circumstances or conditions, without which apneumatosis would not more readily occur in the one than in the other.

These *predisposing circumstances* it may be well to consider a little more closely. Whatever tends to lessen the intensity of the inspiratory effort, and thus to impair its efficiency, will certainly favour the occurrence of Apneumatosis. The introduction of air into the air-cells is the result of a mechanical process, the walls of the chest are separated, and the diameters of the chest increased by the action of certain muscles: the lungs follow the walls of the chest, and increase in bulk, and air is driven in to fill up the vacuum which would otherwise exist within the chest. The principle, indeed, precisely resembles that of the pump. Now, in order that a pump may act efficiently, a

[1] Dr. Stokes.

senting that firmness and resistance which is
the result of this is that at certain situations
during the act of inspiration, and at the cor
lungs little expansion of the pulmonary tissue
of the thoracic walls may sometimes be obser
breathing vigorously when the air-tubes are e
The diaphragm, which is the chief inspirator
also tends to draw in the chest walls at the
which it is attached, if those walls do not prese
rigidity. The point at which the chest walls
is at the junction of the cartilages with the r
more especially exhibit this want of power to
pressure are those just above and below the n
seventh, inclusive. Not unfrequently a gr
passing downwards at the junction of the car
each side, marking the degree to which these
Rickets is a frequent source of this, rendering
than they should be. Sir William Jenner has p
the great influence of rickets in producing thi
cumstance which acts in a somewhat differen
induced general weakness. In this case, the
and draw asunder the ribs are not powerful e
opposing force of the diaphragm; the ribs her
but the muscles are incapable of retaining t
vated, while the diaphragm acts. A combi
here mentioned—viz. deficient rigidity of t
of the thorax, and deficient power of the i
have a very considerable influence in dimin
the inspiratory act.

But under ordinary circumstances nature
these defects. If the chest-walls give wa

Catarrhal inflammation of the air-tubes is generally the origin of the obstruction in question. It produces, in the first place, a swelling of the mucous membrane, and secondly, a secretion of fluid; the one diminishing the calibre of the air-tube, the other obstructing it. Unless the child possess sufficient strength to overcome this obstruction (a strength often wanting) by exercising a greater effort than usual, Apneumatosis of certain parts of the lung will be produced in the manner previously described. The fact that, on the one hand, the small air-tubes are proportionately less in the child than in the adult (Fuchs), and on the other, that bronchial inflammation is so exceedingly common in childhood, will present conditions highly favourable for the production of the lesion, coupled, as they often are, with the partly inherent, defective mechanism of the inspiratory act at this period of life.

The researches of Hutchinson and others have shown that the act of inspiration is one-third less powerful than that of expiration. Under the morbid conditions just pointed out the disadvantage under which the inspiration labours is increased, while the efficiency of the expiratory effort is but little impaired. All the conditions mentioned are such as render the inspiration more difficult, and tend to prevent the passage of air into the air-cells. Inspiration being entirely dependent on muscular effort, is directly influenced by the degree in which that effort can be exercised, subject to certain modifications already pointed out; whilst the expiratory act being in part the result of the reaction of the elastic tissue of the lung, is much less liable to alteration of this kind. This then is another circumstance facilitating the removal of air from the air-cells when the tubes contain an undue quantity of fluid, the obstruction interfering with the inspiratory, but not to a corresponding degree with the expiratory, effort.

A condition which somewhat interferes with the inspiratory act is undue distension of the abdominal cavity, from whatever cause.[1] The diaphragm cannot descend to the full extent necessary, and less air than usual enters the chest. In common with most of the other conditions named this distension of the abdomen will not be effective in the production of Apneumatosis, unless co-existing with obstruction in the air-tubes themselves. The practice which often prevails of binding up the abdomen of the infant tightly must act in precisely the same way, and if the child be attacked with bronchial catarrh it is not difficult to conceive that the mechanism of the inspiratory act may be so impaired, under this combination of evils, as to favour the occurrence of Apneumatosis.

Certain affections of the air-tubes more readily than others produce obstruction and consequent Apneumatosis. Infants having portions of their lungs in a state of atelectasis are more liable to suffer from Apneumatosis than those in whom the lungs have been fully aërated at birth; atelectasis is therefore a predisposing circumstance.

[1] This point has not escaped the notice of Dr. Gairdner (loc. cit.).

Apneumatosis is not by any means frequently observed, in such a degree at least as to prove fatal, after the age of five or six years; it is very common, however, before this period, and in general terms its frequency may be said to be inversely as the age. The first few months of the infant's life are those in which the lung most readily returns to the quasi-fœtal state, loses its gaseous contents, and becomes apneumatic. As the muscular power becomes greater, and the framework of the thorax becomes firmer and more consolidated, Apneumatosis less commonly occurs. The mortality from affections designated in the Registrar-General's reports as pneumonia, hooping-cough, bronchitis, and influenza, in the first year of life, is a rough index of the comparative frequency with which Apneumatosis occurs at this period of life. The result of examination of a large number of cases of children dying from bronchitic and allied affections during the first year of life, was, that with hardly an exception Apneumatosis was present in all, other complications being in many cases also noticed. I am inclined to speak less positively of the state of the lungs present in children dying of such affections after the age of about five years, opportunities being much more rarely afforded of studying the post-mortem changes after this period.

In round numbers the deaths during the first five years of life, and set down in the Registrar-General's Reports under the heads Hooping-cough, Influenza, Bronchitis, and Pneumonia, amount to 25 per cent. of the total mortality at those ages; between the ages of five to ten years, they amount to 10 per cent. of the total mortality; between the ages of ten and fifteen, to 5 per cent. After the second year the mortality from these diseases gradually diminishes: the inference to be drawn is, that the frequency with which Apneumatosis occurs is subject to a corresponding diminution.

The effects produced on the system generally by the presence of Apneumatosis.—Children in whom the lungs are extensively affected with Apneumatosis die of a slow asphyxia, and the manner in which this effect is produced is sufficiently obvious. No respiration, in the mechanical or physiological sense of the word, can take place in the lobules which are collapsed; these portions have become absolutely useless so far as the oxygenation of the blood is concerned; the effect is the same as if the size of the lung had been reduced in a corresponding ratio by complete removal of these portions. It has been shown that the degree to which lobes may be affected is often very considerable in the aggregate; as much as half of the entire lungs has been found to be involved in some cases. The fact that the surface still available for respiration is thus diminished explains the symptoms observed in such cases—the quickened movements of the chest, the distress, and dyspnœa. It is a curious circumstance, and one which of all others should have prevented the older observers from deciding as to the purely inflammatory nature of the lesion in question, that in cases of Apneumatosis a stage soon sets in characterised by great pallidity of the surface, bloodlessness of the integu-

nent, and excessive debility. The surface becomes cold and the decarbonization of the blood is thus shown to be reduced to a minimum. The condition of a child in an advanced state of Apneumatosis in fact bears a great resemblance to that of one of the cold-blooded animals. The asphyxia comes on very slowly and gradually, the system apparently accommodating itself to the lowered respiratory function, less blood circulates through the lung, and less in the system generally. All organs suffer; the energy of the muscles is impaired; they no longer contract with force and vigour. Further portions of the lungs become apneumatic from this very circumstance, and when this has reached its extreme limit the patient dies. In the outset there is more congestion present in the skin, face, &c., but the asphyxia afterwards observed is of a more chronic, and apparently less congestive form.

The circulation is necessarily greatly affected. The blood ceases to circulate in the lobules deprived of air. The cessation does not take place immediately, but after the lapse of a certain time. The first effect of collapse of the air-cells on the circulation in the lobules affected is to retard the flow of blood,—to produce congestion. The blood which at first flows through the part more slowly than usual soon ceases to flow at all. What then becomes of it? Dr. Richardson's experiments have shown that blood will remain for some little time fluid, if preserved from contact with air at rest within the body, but after a time it coagulates. Thus then a second effect, and one occurring later, is coagulation of the blood in the apneumatic lobules. The presence of these clots within the blood-vessels of the lobules, and their various conditions as regards consistence, density, colour, &c., explain the difference observed in individual cases, in the appearance of the section of apneumatic lobules. Fuchs [1] describes after Stilling, the changes which the clot ("der thrombus") found within the vessel undergoes, as follows. At first it lies free within the vessel, but after a time varying in the smaller vessels from two or three days; in the larger, from five to six days, it becomes adherent to the walls of the vessels. Later still it becomes whiter and more dense and contracted, resembling the walls of the vessel in appearance; finally the vessel becomes obliterated, this termination taking place in the small vessels in 20—22 days, in the larger in 30—40 days. The difficulty occasionally experienced in inflating apneumatic lobules is attributed by Fuchs to the contraction which the lung-tissue has undergone as a consequence of the process thus described. The changes which take place in the blood-vessels must after a certain time be an insuperable obstacle to the restoration of the function of the parts involved. An effect of the retardation of the current will be distension of the blood-vessels, and the bulk of the lobules reduced by collapse of the air-cells is still preserved by this distension. Various dynamical effects may thus result. The forcible inspiratory efforts may even produce such distension of the blood-vessels as to render the lobule in question

[1] Loc. cit. p. 75.

larger than usual. This accounts for the increased size of the apneumatic lobules which, as before stated, is sometimes observed. A further remarkable dynamic effect is the unnatural distension of air-cells in other adjacent portions of the lung: emphysema is in fact almost invariably present in cases of Apneumatosis. Large patches of lung present air-vesicles greatly increased in size.

SYMPTOMS.—The symptoms observable in cases of Apneumatosis are quite peculiar, and more reliance can be placed upon them as indicating the presence of the lesion in question than on the physical signs, unless large portions of certain lobules are affected. When the lungs are extensively affected, the state in which the child is found is generally as follows :—There is great prostration and debility, restlessness and inability to sleep. The temperature of the skin and extremities rapidly falls, and the skin is either very pale or of a dusky hue, the lips have a bluish cast, the eyes are sunken, the skin hangs in folds on the attenuated and wasted limbs, and the child appears prematurely aged, having lost the infantine expression peculiar to a healthy child. The pulse is very quick and often hardly to be felt. There is a constant cry, this being of a whining character, and often very feeble. The respiratory function undergoes important changes, manifest in the altered characters observed. The distinctive feature of the respiration is its *shallowness*, it being very evident that very little air enters and escapes from the chest at each successive movement of the walls. The respiratory movements are much quickened; in a child a year old, the number of respirations in a minute may be as high as seventy or even eighty, and if younger than this higher still. The *rhythm* of the movement is altogether changed, being what is called " expiratory," the interval occurring between inspiration and expiration instead of between expiration and inspiration. This is not pathognomonic of the presence of Apneumatosis, for it may be observed in other cases, but it always coexists with the lesion in question. The dyspnœa in fact is extreme, though not accompanied with that degree of lividity of the face and evident distress usually a concomitant of intense dyspnœa. It is evident also that the dyspnœa is not dependent upon pain in the chest, as is the case in pleurisy; the child gives no sign of that kind of suffering which is observed when inflammation of the pleura is present; the suffering is of another character altogether. The cough is very distinctive. In bad cases it can hardly be called a cough at all; the little patient is perpetually making feeble expiratory efforts which produce no effect in evacuating the contents of the tubes, and if the thorax be uncovered, it will be seen that little or no diminution of its bulk takes place during these ineffectual attempts to free the bronchi from the obstructing mucus. These attempts are moreover generally followed by a cry, an expression of impatience at the inadequate result obtained. Nothing can be more significant than the character of the cough, the inefficient nature of which is explained by the fact, that there is a deficiency of air in certain parts of the lungs; for as

already pointed out each lobule is a miniature lung, and the presence air is necessary for the production of that jerking expulsive effect constituting a cough. The dyspnœa present in these cases is usually attributed to the presence of mucus in the tubes, but this is not the whole truth; that mucus would be expelled if there were sufficient air behind it, and the patient had, so to speak, the usual control over that air, and could thus drive it out. The dyspnœa observed in bronchitis alone is of a different character, more suffocative, and more productive of congestion; there is more heat of skin and fever present also; but these febrile symptoms disappear in great part when the lungs become extensively Apneumatic. The *physical examination* of the chest affords information of a very valuable character. The yield-ing nature of the thoracic walls in infancy has been spoken of as pre-disposing to the occurrence of Apneumatosis. That the chest walls do actually give way during life, we have practical proof on watching the movements of the chest during respiration in a child whose lungs are extensively apneumatic. The younger the child the more readily does this take place. During inspiration the lower part of the chest is strongly retracted, and the diameter of the chest diminished at this situation, the converse of what is observed in health. Not only do the firmer parietes of the chest thus fall in, following the tractile influence of the diaphragm, but the intercostal spaces become much more manifest, sinking in during the act of inspiration. Conversely, during expiration the same parts may be seen to move outwards to a slight extent. The retraction of the chest walls during inspiration may be observed when Apneumatosis is not present in consequence of unnatural mobility of the parts, a circumstance previously alluded to, but it is, nevertheless, a sign of considerable importance. The change in the shape and contour of the chest produced by Apneumatosis has been already described.

The results of percussion and auscultation in the young child are in all cases less to be depended on than in the case of the adult. Where the Apneumatosis is extensive, the percussion sound is dull and attended with some degree of resistance; but as it generally happens that the lobules affected are more or less intermixed with others which are healthy, or which even contain a greater amount of air than usual, this dulness or percussion often escapes detection in cases when the aggregate amount of Apneumatosis is considerable. Emphysema, as before stated, is constantly combined with Apneumatosis. The pre-sence of these emphysematous patches will interfere with the results of percussion practised immediately over them in a manner sufficiently obvious. When the whole of one lobe is affected, or when, as it frequently occurred in cases coming under our own observations, the greater part of the lower lobe on either side has lost its gaseous contents, the dulness on percussion has been very marked, and the width of the surface presenting this dulness has increased from day to day under observation. Generally speaking, then, the presence of dulness on percussion is a positive sign, but its absence is, for the

reasons just stated, not a negative one. It is to be looked for at the basis of the chest posteriorly, and next in order of frequency at the same position anteriorly.

The respiratory murmur disappears over those parts of lung affected with Apneumatosis, if the disease be widely spread. On the whole, however, it is rare to meet with entire absence of respiratory sound on auscultation, some sounds being still transmitted from deeper parts. We have observed its complete absence more especially in the case of very young infants. The more usual circumstance is that the breath-sound is, when not masked by rhonchi, somewhat bronchial in character, the solidified lung transmitting the sound from the larger air-tubes. It is somewhat rare, however, to meet with cases in which rhonchi, due to the passage of air through mucus, are not audible. With reference to these rhonchi, the most striking character they possess is a degree of coarseness and roughness, not often noticed in the case of the adult. Rhonchal fremitus is only present in the early stage. The true crepitant rhonchus, which is in the adult the chief distinctive sign of the presence of pneumonia, is not heard. Authors have generally accounted for the absence of this pneumonic crepitus in young children, supposed by them to be the subject of "pneumonia," by concluding that the peculiarities of the structure of the child's lung prevented its development; but the fact is, there being no pneumonia, there is, therefore, no crepitus. It is unnecessary further to describe the various kinds of rhonchi which are found to be present in these cases. They depend on the bronchitis present. An important circumstance is the rapidity with which these changes from the normal condition may take place. A large surface of the lung may become solid, causing dulness on percussion and loss of respiratory murmur in twenty-four hours; the limits within which the alterations are observed may also change in as short a time as this. Valleix observes that a dulness of all the posterior part of the right and of the lower third of the posterior surface of the chest may supervene in the space of twenty-nine hours, no sign of this dulness having been present the day before.[1] This is, perhaps, more especially the case in very young infants, for in older children the lung requires to be longer subjected to the necessary process in order that large portions may become apneumatic. The changeableness of the character of the sounds conveyed to the ear by the stethoscope, is of course produced by and follows the alterations in the lung-tissue here alluded to.

The peculiarity of the child's voice interferes with any observations on the intensity of the resonance as felt by the hand, the vocal fremitus.

Such are the symptoms and signs observed in cases when the Apneumatosis is tolerably extensive and well marked. In cases where it is inconsiderable in amount, and scattered over different parts of the lobes, the physical signs may be wholly inadequate to determine its presence, and the general symptoms then afford more information.

[1] Loc. cit. p. 128.

Cases, indeed, not unfrequently occur in which death having taken place, the Apneumatosis is found to be considerable, but having the characters here alluded to, no dulness on percussion, no positive sign of solidification having been detected during life.

The *course, duration,* and *mode of termination* of the disease must necessarily vary in different cases. The disease is generally fatal, when involving the lungs to a considerable degree. A child, badly fed, living in a close, confined apartment, breathing constantly a vitiated air, may, if attacked by bronchitis, die in consequence of the Apneumatosis resulting therefrom, in a short space of time, but the time will vary in different cases. If the child be affected with atelectasis to begin with, the disease is more quickly fatal, but if previously strong and tolerably healthy, its duration is proportionately prolonged. Hooping-cough is exceedingly fatal to very young children, because the bronchitis which accompanies it so readily gives rise to Apneumatosis;[1] but it is well known that it is amongst the children of the poorer classes only that the disease occasions so great a mortality, where, in fact, the predisposing causes before alluded to are allowed to come into operation. The hygienic conditions being favourable, Apneumatosis both less readily occurs, and, when produced, is less likely to prove fatal, than when this is not the case. Unless interfered with, the natural course of the malady is from bad to worse: from the nature of things, the disease tends to intensify itself, and from day to day the affection increases by involving more of the lung substance. As the disease extends, the patient becomes very feeble, unable to cough, or expel the mucus from the tubes, and the quantity of blood in the system seems to undergo a diminution. This is proved by the result of post-mortem examination in chronic cases, and is made evident during life by the pallid, bleached appearance of the patient. After suffering under the symptoms for, it may be, two or three weeks, the death takes place by what is, in reality, a slow asphyxia. The course of the disease may be more rapid, as is sometimes the case in infants who have previously enjoyed a better state of health. These are seized with a severe attack of bronchitis, pervading the smaller as well as the larger tubes, and large portions of the lungs suddenly, or comparatively so at least, become apneumatic and deeply congested; death then rapidly supervenes, the asphyxia being more suffocative and acute in character than in the former case. In both cases, recovery may of course be the result, although the lungs are a long time before their functional activity is completely restored; the seeds of future mischief are some of them left behind, and may subsequently induce a return of the disease: chronic emphysema is a very frequent result of Apneumatosis.

That large portions of lung substance may, within a very short space of time, return to the healthy state, which a short time before

[1] See the author's essay "On Pathology of Hooping-cough" (Churchill, 1855), containing the results of the examination of the lungs after death in nineteen fatal cases of this disease.

had been obviously apneumatic, has been with us matter of observa-
tion, and the same circumstance has been noticed by others. The
effect of judicious treatment, in restoring clearness of percussion
sound and respiratory murmur, is occasionally indeed very marked,
and is of itself a sufficient evidence that the dulness which before
existed was not due to true pneumonic consolidation of the lung.
The cure is often impeded, may often be prevented by the emphysema
which coexists; for although the child may have the power of inspir-
ing forcibly restored, the thorax being already filled by the emphyse-
matous distension of certain of the air-cells, no expansion of the
apneumatic lobules occurs.

The PROGNOSIS, in a particular case, is favourable if the affection be
recent, occurring in a tolerably healthy child, and when the muscular
power is not greatly reduced: the hygienic and other conditions in
which the patient may be placed, are very important features in the
case, as regards the prognosis. In infants, Apneumatosis occurring in
connexion with hooping-cough is especially fatal; few recover from it
when placed, as are the children of the lower orders in large towns,
under unfavourable hygienic conditions.

DIAGNOSIS.—Dulness on percussion and bronchial respiration are
of most value, where they are present; under other circumstances the
altered character of the respiratory movements, the retraction of the
chest walls, combined with the general condition of the patient, and
the history of the case, are data on which a diagnosis may be arrived
at with tolerable facility.

The diagnostic signs of atelectasis cannot be entered on here. In
reference to the other conditions with which Apneumatosis may be
confounded, and which it is necessary therefore to distinguish, a few
remarks will suffice. *True pneumonia* is very rare in early infancy;
the presumption in a particular case will be, therefore, that this condi-
tion is not present. The absence of the continued and persistent heat
of skin, the absence of the pneumonic crepitus, afford negative evidence
tending to the same conclusion. It will be more difficult, however,
to distinguish between a case of true pneumonia, in which the inflam-
matory acute stage has passed away, leaving consolidation of the
lung, and one in which Apneumatosis is present. Another condition
—*extensive deposit of miliary tubercle* in the substance of the lung—
might present symptoms and physical signs somewhat resembling
those observed in the case of Apneumatosis. The history of the case
would, however, show that symptoms, as cough, wasting, &c., had
been observed for some time previously; and the general condition of
the patient, together with this circumstance, could hardly fail to lead
to a correct conclusion as to the nature of the case. It may be
remarked, however, by the way, that Apneumatosis, as a complication,
is often discovered after death in cases of tubercular disease of the
lungs. In cases of *pleurisy*, with effusion, there would be dulness on

percussion over the lower part of the base of the thorax, together with absence of breath-sound on auscultation, both of which physical signs are present in cases of Apneumatosis: it is to be distinguished from the latter condition, by the greater intensity and width of the dulness on percussion, by the more complete absence of respiratory murmur, observed in the former case. Moreover, in cases of Apneumatosis, it is generally found that the dulness is not limited to one side, as is more frequently the case in pleurisy.

TREATMENT.—Patients affected with Apneumatosis have lost for all functional purposes large portions of the lungs; it is our business to endeavour to restore these portions to their functional activity, and to prevent others from falling into a similar condition. Clear indications for treatment will be found on examining the class of causes, effective in the production of Apneumatosis. As every circumstance which tends to lower the muscular and vital power of the patient favours the production of Apneumatosis, it is very obvious that we are not likely to improve matters by the exhibition of medicines having a lowering character, or by the abstraction of blood, in a case where the child is already too feeble. Setting aside for a moment the consideration of the bronchitis itself, which is or has been present in a particular case, there seems to be no good reason for the employment of depletive or depressing remedies in the treatment of Apneumatosis. There are many reasons against this procedure. The older observers carried their principles into practice: they considered that they had to treat pneumonia, and they treated it accordingly. It is no less incumbent on us to adopt a treatment precisely the reverse.

We are decidedly of opinion that, as a general rule, when an infant is the subject of Apneumatosis, depletion, local or otherwise, is not admissible. The same must be said of the internal administration of tartar-emetic in repeated doses.

One of the chief difficulties to be encountered is the impediment offered to the entry of air, by the presence of mucus, which the child is unable to expel. A primary object is then to assist the respiratory efforts of the patient, at the same time that we endeavour to diminish the excessive secretion of mucus in the air-tubes. Counter-irritation is a valuable means to this end, the degree of which must be adapted to the strength of the patient and the duration of the disease. Mustard poultices are very useful; they can be frequently repeated, and do not produce prostration. Blisters are objectionable from their weakening tendency. We have found frictions of the chest to be followed by markedly good effects, when performed in the following manner:—The hand, lubricated with sweet oil, is to be rubbed tolerably briskly over the whole surface of the chest for ten minutes or a quarter of an hour together, two or three times a day. The result obtained is twofold, a counter-irritant effect is produced, the blood being drawn to the surface and the internal congestion thus diminished, and the movements of the chest are very much facilitated.

removing the contents of the air-tubes than a...
pose ipecacuanha seems to be the best; eigh...
powder is a proper dose for an infant a year o...
as is the emetic in question, it is not to be admir...
certain circumstances. If the patient be very...
of some days' duration, the emetic may be uns...
indicated, it may be given once, but is not to...
efficiently, the object in view is attained, and m...
its repetition unless after the lapse of a certain...
anha wine (about ten drops), given in a little...
hours, has the effect of promoting expectora...
bowels must not be neglected, but mild aperie...
The food must be extremly simple, but at the...
The breast milk for an infant, milk and wate...
quite sufficient in ordinary cases. The case is,...
the lungs are extensively affected. Then al...
directed to the maintenance of the vital po...
safe, even mild expectorants may be improper...
matic spirit of ammonia, or steel wine, or at a...
of the phosphate of iron, must be given, and...
diet a little port-wine and water, or brandy-an...
tea. In dieting young children it is too often...
trated food is not well digested, and rich crea...
in many cases act as irritant poisons if take...
an infant; great care must be taken to dil...
that it may be easily digested, or it will do...
than good.

BRONCHITIS.

By Frederick T. Roberts, M.D. Lond.

DEFINITION.—An affection of the mucous membrane lining the bronchial tubes, varying from mere hyperæmia of limited extent, to an intense and widely-distributed inflammation, which may involve the deeper structures. It usually gives rise to an increased and altered secretion, containing abundant cells, but in some cases a plastic exudation is thrown out into the tubes. Hence there are two chief forms of Bronchitis, named the Catarrhal and Plastic or Croupous, each occurring as an acute and chronic affection.

SYNONYMS.—Bronchial Catarrh; Catarrhus Pituitosus; Catarrhus Suffocativus; Angina Bronchialis; Erysipelas Pulmonis; Peripneumonia Notha; Bronchite (French); Bronchialentzundung (German).

ACUTE CATARRHAL BRONCHITIS. ACUTE BRONCHIAL CATARRH.

NATURAL HISTORY.—CAUSES. — I. *Predisposing.* — These are due partly to the individual, partly to surrounding external conditions. The following include the most important :—

1. *Age.*—There is no age at which Bronchitis does not occur, but it is far more commonly met with at the extremes of life. It is a very frequent complaint among children, especially during the first two years of life, while dentition is going on, and persons of advanced years are also exceedingly subject to it. The occurrence in children of various affections which tend to have Bronchitis as a complication, and in old persons, of chronic pulmonary, cardiac, and other diseases, will to some extent account for this; while, in addition, they possess less vital power to resist the ordinary exciting causes. The table on page 884 shows the rate of mortality at the various ages, during the year 1868, as contained in the Registrar-General's Reports, but it only gives an approximate idea of the relative frequency, as Bronchitis is so much more fatal among the old and young.

MALES.

Under one year	3,849	Ten years	40	Fifty-five years . 2,430
One year	1,585	Fifteen years	52	Sixty-five years . 3,002
Two years	562	Twenty years	85	Seventy-five years 1,956
Three years	289	Twenty-five years	331	Eighty-five years 300
Four years	139	Thirty-five years	728	Above 10
Five years	207	Forty-five years . 1,369		

FEMALES.

Under one year	2,969	Ten years	47	Fifty-five years . 2,316
One year	1,585	Fifteen years	61	Sixty-five years . 3,218
Two years	594	Twenty-years	93	Seventy-five years 2,158
Three years	243	Twenty-five years	327	Eighty-five years 440
Four years	161	Thirty-five years	615	Above 20
Five years	211	Forty-five years . 1,267		

2. *Sex* does not seem to influence the number of cases materially. In the year 1868, 16,934 deaths were recorded among males, as compared with 16,324 among females; and it will be seen from the tables that the first year of life gives the greatest difference. Probably men have bronchitic attacks more frequently than women during the adult years, being more exposed to cold, &c.

3. *Habits.*—Unquestionably those who indulge in luxurious and enervating habits, and who wrap themselves immoderately, or live in rooms of a high temperature, produce a relaxing and depressing effect upon the system, and render themselves more obnoxious to slight external influences. The excessive care which many children receive in these respects is certainly injurious; while, on the other hand, their resisting power may be increased by a judicious process of inuring them to various atmospheric changes.

4. *Temperament.*—It is said that those of a sanguineous and lymphatic temperament are more liable to be attacked, but I am not aware of any positive facts bearing out this statement.

5. *State of General Health.*—A constitutionally weak state of the system, or debility resulting from any cause, such as deficient and improper food, or severe illness, predisposes to Bronchitis; while the existence of any positive constitutional disease, such as tuberculosis, rickets, Bright's disease, gout, diabetes, cancer, &c., is still more favourable for its occurrence.

6. *Condition of the Lungs and Bronchi.*—The presence of any deposit in connexion with the lungs, as tubercle or cancer, as well as the existence of certain chronic affections, especially emphysema and dilated bronchi, necessarily favours the setting-up of Bronchitis. If the mucous membrane has been once attacked, it is rendered more susceptible, and this susceptibility is increased with each attack; hence it is not at all uncommon for a person to suffer every year when the cold weather sets in.

7. *State of the Heart and Circulation.*—Any heart disease that interferes with the return of the blood through the bronchial veins, or anything that causes extra pressure upon the circulation in the bronchial arteries, has a considerable predisposing influence as regards catarrh, and may even excite it. In the manner last mentioned, abundant ascites is said to act by exerting pressure upon the aorta below the origin of the bronchial arteries, and thus throwing an extra strain upon them.

8. *Occupation.*—The occupations which seem to be specially favourable to Bronchitis, are those which involve much exposure to cold and wet, or sudden and marked changes of temperature, and those which lead to the inhalation of irritating particles floating in the atmosphere, such as cotton, steel, charcoal, &c.

9. *Social Position.*—Those among the poorer ranks of society are, for several reasons, very liable to Bronchitis. A large number of cases occur among hospital and dispensary patients.

10. *Climate.*—Bronchitis is very much more common in climates characterised by considerable moisture of the atmosphere, combined with low temperature; and especially where there are sudden and marked variations in temperature. The same observation applies to individual districts; those that are bleak and damp being rarely free from bronchitic cases. It is an exceedingly prevalent disease in this country, and stands very high as a cause of death. In 1867, 40,373 deaths occurred from Bronchitis, being in the proportion of 1,902 to every million persons living, and of 86,554 in every million deaths. In 1868, the number of deaths was 33,258, giving a proportion of 69,765 per million deaths. The mean rate of mortality for 15 years, from 1850 to 1864, was 1,344·4 in every million living. It occurs in different districts with very variable frequency. The following statistical summing up gives, approximately, the proportion of deaths from Bronchitis to the number of inhabitants in the different districts during the year 1868 :—

London, 1 in 442·3; South Eastern Counties, 1 in 805·01; South Midland Counties, 1 in 834·7; Eastern Counties, 1 in 987·5; South Western Counties, 1 in 844·8; West Midland Counties, 1 in 665·03; North Midland Counties, 1 in 876·2; North Western Counties (Cheshire and Lancashire), 1 in 379·5; Yorkshire, 1 in 541·5; Northern Counties, 1 in 774·8; Monmouthshire and Wales, 1 in 955·4.

11. The foregoing statistics prove that Bronchitis is much more prevalent in large towns and cities than in country places, and the reasons for this will be obvious. The same remark applies to the fact that the poorer districts of cities and towns furnish by far the greater number of cases. Places where extensive manufactures are carried on, loading the atmosphere with various irritating materials, have also always a considerable proportion of cases.

12. *Season.*—By far the largest number of cases is met with during the colder months of the year, extending usually from the end of

influence.

II. *Exciting.*—1. In the great majority of
form or other, acts as the immediate exciting
chitis. It may produce its effects in various
may arise from the breathing of cold air, es
time loaded with moisture, and particularly if
change from a warm and dry atmosphere; eme
into a cold atmosphere, particularly when in
and sitting in a cold draught, contribute nume
insufficient amount of clothing in cold wea
upper part of the body; neglecting to change
wet feet; sleeping in damp beds, &c., are all fr
who drivel constantly and profusely, so that
the chest are always moist, are said to be ver
In most of the instances where the cause
probable that the patient has " taken cold " in
modes in which cold produces its injurious e
by causing local irritation of the bronchial
disturbing its circulation and nutrition; secon
system at large in some way or other not un
being only a part of a general disturbance.

2. On the other hand, sudden great heat aft
the night air into a very hot room, is said so
chitis, but this is difficult to substantiate.

3. Another important exciting cause is the
irritants upon the mucous membrane lining
may arise from certain conditions of the atmos
very high or low temperature, or from its con
or vapour, *e.g.* sulphurous anhydride, chlori
having certain minute particles floating in
filings, charcoal, cotton, flour, &c., and in the

fever, &c. It is particularly liable to occur in the eruptive fevers, if the eruption comes out imperfectly, or suddenly recedes. Neglect of proper precautions during convalescence from these affections, is very apt to lead to dangerous Bronchitis. The poison of syphilis, as well as that of gout and rheumatism, also produces this affection, and it is particularly prone to occur in the last two diseases if sudden metastasis takes place. The state of the blood must also account for those cases that are said to result from the rapid disappearance of the eruption of erysipelas, the suppression of long-continued discharges, whether natural or morbid, and the too rapid cure of an old-standing skin disease. Iodine taken internally sometimes causes bronchial catarrh, evidently due to its presence in the blood.

5. Various deposits in the lung may not only predispose to, but actually excite inflammation of the mucous membrane. It is constantly met with more or less when tubercle or cancer is present, and is then prone to be localized.

6. In connexion with influenza, Bronchitis occurs epidemically, without our being able to trace it to any special cause. At certain times of the year a large number of persons are often simultaneously attacked, so that the complaint may almost be said to be epidemic, but this is due to obvious atmospheric conditions already alluded to.

SYMPTOMATOLOGY.—The clinical history of Acute Bronchitis varies considerably under different circumstances, and an attack may range from a slight "cold in the chest," to one inducing suffocation and gravely affecting the system at large. The chief reasons for these variations are to be found in the age, general condition, and health of the patient, the previous state of the lungs, the extent of mucous membrane involved, and the immediate cause of the disease.

In practice, the following forms are met with :—

I. Acute Primary or Idiopathic Bronchitis, the result of "cold," there being no previous evident lung affection :—

   1. Involving the larger and middle-sized tubes only, and not extending into the smaller tubes.
   2. Implicating the smaller tubes—"Capillary Bronchitis."

II. Secondary Bronchitis :—

   1. In connexion with the exanthemata.
   2. In certain blood-diseases.
   3. After chronic lung and heart affections.

III. Mechanical :—

   1. Hay-asthma, &c.
   2. That resulting from mineral and other irritant particles.

IV. Epidemic.

The primary forms it will be necessary to describe at some length, but the others will call for only a few remarks, pointing out in what respects they differ: whereas Epidemic Bronchitis it will not be requisite to allude to again, as it belongs to Influenza.

1. ACUTE IDIOPATHIC BRONCHITIS, not extending beyond the middle-sized tubes.

Invasion.—This is almost always characterised by the occurrence of symptoms of so-called "catarrh," in consequence of the mucous membrane lining the nasal cavities and their communicating sinuses being affected, and, frequently, the conjunctivæ. There is an irritating watery flow from the nose and eyes, and a feeling of fulness, heat, and soreness in these parts, with frequent sneezing fits. Frontal headache exists, due to the state of the frontal sinuses. The upper and back part of the throat often feels sore and rough, and frequent attempts are made to clear it from mucus. There is generally uneasiness over the larynx, and the voice is more or less hoarse and husky, indicating that the mucous membrane here is also implicated. Not uncommonly the catarrh seems to spread regularly downwards along the respiratory tract, beginning in the nose. In some instances the larynx is alone involved at first, while in others the bronchial mucous membrane seems to suffer from the outset, the upper part of the tract escaping; but this rarely happens in the form now under consideration. Along with these local symptoms there are others of a general character, almost always present more or less. The patient feels chilly, or there may be even rigors in a sensitive person, but they are never of marked intensity, and several occur at irregular intervals, not a single prolonged fit of shivering. Their severity is usually in proportion to the extent of the inflammation. In the intervals between them the patient feels hot, but the temperature is not raised, as evidenced by the thermometer, or only slightly. The pulse is often somewhat increased in frequency. The limbs and joints, or even the body generally, are affected with pains of an aching, contused character, and there is a general sense of fatigue, languor, and want of energy, the patient experiencing a disinclination for any occupation, mental or physical. He is heavy and drowsy, but sleep is often restless and uneasy. There is frequently a furred tongue, anorexia, and constipation, evidencing that the alimentary canal also suffers. In nervous, irritable persons, and in the older children, slight delirium is said to be present sometimes; while in younger children, especially during the period of dentition, and in those who are weakly, a fit of convulsions may usher in the attack.

After the initiatory symptoms have lasted a brief but variable time, those characteristic of the bronchial inflammation set in. They may be very slight, or tolerably severe, and are "local" and "general."

Local.—Various unpleasant or painful sensations are experienced behind the sternum, especially towards its upper part, and in the supra-sternal notch. These are, more or less heat, sometimes reaching to actual burning, and a sense of soreness or rawness, which may amount

to considerable pain—as a rule, however, it is not severe, when the patient is quiet. A deep inspiration aggravates these feelings in a variable degree, while the act of coughing gives rise to much positive pain, of a raw, aching, burning, or tearing character. This is not only complained of behind the sternum, but also radiates towards the sides, as if in the course of the primary bronchial divisions. If the cough is severe and frequent, a feeling of soreness or aching is soon felt all over the chest, but especially towards its sides, and at the base where the abdominal muscles are attached. A very unpleasant irritation or tickling is also experienced above and behind the sternum, which excites the cough. Tenderness over the sternum is often present, the skin feeling sore on percussion. These sensations vary much in intensity, and may merely amount to a diffused feeling of slight heat and uneasiness over the front of the chest, but most marked behind the sternum.

Dyspnœa is not a prominent symptom, but the frequency of the respirations is often somewhat increased, and the pulse-respiration ratio may be more or less altered. The act of breathing is laboured in many cases, and there is always a sense of oppression, weight, and tightness about the chest, especially towards its upper part.

Cough is one of the earliest and most striking symptoms. It is loud, and usually a little hoarse at first, owing to the larynx being affected; otherwise it is free from hoarseness. It comes on in paroxysms, either spontaneously, or from any slight irritation, as inhaling cold air. These last a variable time, and cannot be suppressed. They increase in frequency as the disease advances, and often become very violent, especially after a sleep, and on first lying down at night. There is no expectoration at the outset, the cough being hard and dry, but afterwards each fit ends with expectoration. It is evidently due at first to the abnormally irritable condition of the mucous membrane, and subsequently to the presence of excessive and altered secretion in contact with it, which is itself probably of an irritating nature at first. The *expectoration* varies in its characters at different periods of the case. At the beginning it is small in quantity, thin and watery in appearance, almost transparent, but frothy, and has a saltish taste. The changes it undergoes are : increase in quantity to a variable degree; diminution in transparency, becoming at last almost or quite opaque; increase in consistence and viscidity ; diminution in frothiness ; loss of taste ; and change in colours. Thus, it generally passes through stages of viscid, semi-transparent, slightly yellowish or greyish, frothy mucus, to a muco-purulent or purulent-looking substance, nearly opaque, of a greyish yellow, yellowish, or a greenish yellow colour, and but slightly aërated. It usually runs together into one mass, but a distinct, nummulated form of sputum is sometimes met with, which is thoroughly opaque. Its tenacity and adhesiveness may be so great as to make it stick closely to the vessel containing it, and to admit of its being drawn out into threads. Sometimes it is quite ropy and gelatinous. A few streaks of blood may be seen, especially at the early period. Should an extension of the inflamma-

tion take place, this is indicated by the expectoration once more assuming its early characters in part. As the sputa become altered they are more easily expelled, especially from the larger tubes, and hence the cough abates and is much less painful. *Microscopial characters.*—In the early stage pavement, columnar, and ciliated epithelial cells are seen, with a few imperfectly formed cells. Later there are abundant young cells, discharged from the surface of the mucous membrane, many resembling the so-called exudation corpuscles, and at last pus cells. Molecular and granular matter is seen in quantity; a few blood discs may be present, and occasionally amorphous, fibrinous coagula. Crystals of oxalates, &c., are sometimes visible.

General.—In the slighter cases there are no notable signs of general indisposition, but if the attack is at all severe, the system gives indications of being affected. More or less febrile reaction occurs, the pulse becoming frequent, but rarely above 100 ; at the same time in a healthy person being strong and full. The skin feels hot, but not acridly ; and it may soon be moist. The actual temperature is never very high, but it follows the ordinary rule of increasing in the evening. If the fever precedes the bronchitic symptoms, it is said to be notably more severe. Slight rigors may continue throughout the attack. The tongue is generally more or less furred, but moist; and there is some thirst, with loss of appetite. The bowels are mostly confined. Vomiting may occur, especially after a severe fit of coughing. The urine presents the ordinary febrile characters in a varying degree: the urea and pigments are increased, but the chloride of sodium may be notably diminished. There may be heat during micturition, probably from slight catarrh of the urethral mucous membrane. A sense of languor and weakness continues throughout the case, and there may be considerable depression, quite independent of, or out of proportion to, the febrile state.

A favourable case of this description may run its course in three or five days, or may last two or three weeks, according to the number and size of tubes involved, the depth of the inflammation and the state of the patient. The fever, if any existed, soon abates and the local symptoms gradually subside; the cough, however, often holding on for some time, especially in the mornings, on account of the secretions having accumulated. These cases do not always end in recovery. In very old patients, and in those weakened by disease or want, fever of an adynamic type is apt to be present from the first, or to follow sthenic fever, especially if this has been severe. Then there is great debility, a quick, feeble pulse, a dry, brown tongue, and low delirium. Or it may happen that the patient is unable to expel the secretion formed in the tubes, which therefore collects and tends to pass into the smaller tubes, thus possibly causing inflammation in them, or blocking them up, and leading to slow suffocation. In young infants, even a very little bronchial catarrh may lead to serious results, especially if they are feeble and ill-nourished, or are the sub-

jects of rickets. They are unable to expectorate, and thus the fluids accumulate, and a large tube, or a number of tubes, become blocked up, collapse of portions of the lung resulting from this. Under any of these circumstances a fatal result may ensue. In a comparatively few instances this form of Bronchitis remains as a chronic affection, particularly if it implicates the deeper structures of the tubes.

2. ACUTE BRONCHITIS, involving the minute tubes. *Capillary Bronchitis.*—This is a very dangerous condition, even in a healthy and robust adult; but it is peculiarly grave when children, old people, or very debilitated persons are the subjects of it, among whom it occurs with considerable frequency, in the order in which they are mentioned. This results partly from the great interference with the blood-aëration that it involves, partly from the accompanying fever, which has a strong tendency to become adynamic. In the majority of cases it is preceded by symptoms of inflammation in the larger tubes, or the whole tract may be more or less involved simultaneously or very rapidly. In some instances the smaller tubes seem to be alone affected from the first. The early symptoms may be those already described, or well-marked rigors, severe headache, and sickness may usher in the disease. There may be only slight or very considerable pain behind the sternum, but it is absent if the capillary tubes are alone implicated. Children and aged persons often do not appear to suffer any particular pain. There is always, however, much aching and soreness about the base of the chest and epigastrium, owing to the severe spasmodic contractions of the expiratory muscles during the fits of coughing. This is aggravated during each paroxysm, and patients frequently sit up or bend forwards while they cough, in order to release their abdominal muscles, at the same time pressing their sides, so as to give them support. *Dyspnœa* always attracts attention, but its degree varies materially. It may be limited to accelerated and somewhat laborious breathing, with a feeling of constriction and oppression across the chest; or the respirations may be extremely frequent and hurried, attended with violent efforts during inspiration, and an urgent craving for air. There may be constant or paroxysmal orthopnœa, the latter supposed to be due either to spasm of the bronchial tubes, or to the sudden blocking-up of a large tube with secretion. The absolute frequency of the respirations may rise to 50 or more, and being increased out of proportion to the pulse, the normal ratio is disturbed, being sometimes 2·5 to 1. Wheezing and whistling sounds are often present, audible at a distance, and attending both inspiration and expiration. *Cough* occurs almost continuously, but it also comes on in extremely violent, prolonged, and distressing paroxysms, during which the face becomes turgidly red or purple, the veins swell, and the arteries throb. *Expectoration* is effected with much difficulty, owing to the secretion being exceedingly tenacious and sticky, and having to be expelled from the smaller tubes, while the muscular fibres of the bronchi, which

normally assist expectoration, are probably paralysed in many cases.
The sputa are scanty at first, but soon increase greatly in quantity,
becoming chiefly muco-purulent, yellowish-green, or bright green and
opaque ; or extremely viscid, glutinous, and ropy : they may partly
retain the form of the smaller tubes, and minute cylindrical casts,
consisting of fibrinous exudation, may be present, or irregular particles
of the same substance. Some frothy, lighter mucus from the larger
tubes is mixed, more or less, with the above. Children do not expecto-
rate, or rather they swallow what they bring up, but some of it may
be obtained for examination by wiping the base of the tongue with a
handkerchief after a fit of coughing.

The *constitutional* symptoms are always severe. At first there is
ordinarily considerable fever, which, in the case of healthy adults and
plethoric children, is of the sthenic type, but in the aged and feeble
is prone to be asthenic from the outset, or speedily to assume this
character. The pulse is frequent, quick, and generally full. The skin
is hot, but may be dry or moist. The temperature may reach 103·5
Fahr. in the evening, when it is often 2° in excess of the morning.
Flushing of the face, and headache, increased by the cough, are com-
monly present. Pains are complained of in the trunk and limbs, and
there is a feeling of great weakness and exhaustion. Wasting occurs
in proportion to the fever and to the interference with sleep, which is
generally great. Loaded tongue, anorexia, constipation, are usually
marked symptoms, and there may be much sickness. The urine, in
addition to being febrile, is sometimes slightly albuminous temporarily,
and it is said a trace of sugar is occasionally present. Chloride of
sodium may be almost totally deficient.

The symptoms, both local and general, may, after reaching a
certain point, subside, and gradual recovery take place ; but in the
majority of cases this favourable result does not occur. Indications
of more or less imperfect aëration of the blood are observed in
almost every instance, owing to the impaired respiratory process ;
but in many, especially children, this constitutes the main source of
danger, and leads to a fatal issue. Gradual suffocation is brought
about, and the blood becomes charged with carbonic anhydride, while
its oxygen is proportionately deficient ; and hence the various organs
essential to life are supplied with blood which cannot maintain their
functions. When this happens the face assumes at first a turgid,
bloated, and more or less red, dusky, or livid appearance, but it soon be-
comes generally pale, while the lips, tip of the nose, malar prominences,
and external ears deepen in their lividity, which contrasts strongly
with the surrounding pallor. The veins of the head and neck swell.
The surface generally is also cyanotic in a variable degree, particularly
the fingers and toes, this appearance being very marked under the
nails. The feet and hands may swell from œdema, which may extend
even to the trunk. The temperature rapidly falls, especially that of
the extremities. Cold, clammy sweats break out about the face and
upper part of the body, and then spread universally. Rapid exhaus-

tion of the vital powers follows, and the patient allows his head to sink on the pillow or droop in any direction. The pulse becomes greatly accelerated, weak, small, and compressible, and at last often irregular. Intense thirst is complained of. Cerebral symptoms set in early; the mind wanders, and in many cases a persistent desire to get out of bed is manifested. I have seen this well marked in some adult cases. There is at first perpetual restlessness, with a deeply anxious expression of countenance, and great dread; but these conditions soon change, and the patient becomes more and more indifferent, with dull and heavy eyes, then falling into a drowsy state, out of which for a while he starts suddenly, but which gradually deepens into permanent stupor, and finally complete coma, which precedes death. Convulsions may occur before the final coma. The cough ceases after a time, the power as well as the desire of expectorating being lost. Breathing becomes much quieter, but very hurried and shallow. As a consequence, the secretions gradually fill up the air-tubes, and thus are produced rhonchal sounds, audible at some distance, which change into gurgling as the fluids rise into the larger tubes. The expired air is cool.

The urine is greatly diminished in quantity, and may be totally suppressed.

Death sometimes occurs suddenly, before the brain is much involved, owing to the blocking up of a large bronchus with secretion; which is most liable to happen in young children.

Instead of the symptoms just described, those characteristic of adynamia may arise, especially in the aged or feeble, and where the fever has been excessive. The tissues are rapidly consumed, and the blood loaded with the resulting impurities. The tongue becomes dry and brownish, with a red tip and margins, or a thick dark fur may form upon it behind. The pulse is very frequent and small, often irregular and uncountable. Low, wandering delirium sets in, succeeded by coma. Profuse, clammy sweats break out, the extremities becoming cold. There are no marked cyanotic symptoms at first, but owing to the condition of the sensorium the need of expectoration is not felt, and thus the secretions accumulate in the tubes, this being aided by paralysis of the muscular fibres in the walls of the bronchi, which finally leads to slow suffocation.

In many fatal cases, the two classes of symptoms above described appear to be combined more or less. Certain complications may occur greatly increasing the danger, the chief being lobular or more extensive pulmonary collapse, acute emphysema, lobular or lobar pneumonia, congestion ending in œdema, and pleurisy.

The term "Peripneumonia Notha" is applied rather vaguely to some cases of Bronchitis. With some it is synonymous with Capillary Bronchitis; but it seems more appropriately to refer to the disease occurring in an old or enfeebled subject, after some chronic malady, with febrile symptoms at first, but signs of adynamia, and deficient aëration of the blood setting in early.

3. BRONCHITIS OCCURRING IN CONNEXION WITH THE EXANTHE-MATA.—Some of these are never free from a certain amount of bronchial catarrh, more especially typhoid fever and measles, and it may constitute the chief source of danger. It is very apt to come on insidiously without pain or difficulty of breathing, and scarcely any notable cough or expectoration. In short, physical signs may alone indicate the existence of the catarrh. On the other hand, the attack may be exceedingly severe, and mask for a time the nature of the fever. In measles constantly, and in scarlatina usually, coryza exists at the outset, but in the other fevers it is commonly absent. The Bronchitis may come on early or late in the case. Should it be extensive, or the patient be much weakened, it is a serious complication, and may rapidly lead to a fatal result. It is important to bear in mind the non-occurrence of subjective symptoms, and that it is necessary to employ physical examination of the chest at frequent intervals.

4. BRONCHITIS IN CONNEXION WITH BLOOD DISEASES.—In some instances it may be considered as truly secondary, depending immediately upon the poisoned state of the blood; but in others this only acts as a strong predisposing cause. Here again the disease is prone to come on insidiously, without any marked symptoms, and also to last a long time, often becoming chronic. The expectorated matters are said to contain some of the poisonous materials which accumulate in the blood, such as sugar in diabetes, urea in Bright's disease, uric acid in gout, &c.

5. BRONCHITIS IN CONNEXION WITH CHRONIC LUNG AND HEART DISEASES.—When occurring as the result of deposits in the lungs especially tubercle, Bronchitis is very commonly localized to their immediate neighbourhood, and hence is often confined to the apex It is not preceded by coryza, and there are usually no marked symptoms. Should there have been previous chronic Bronchitis, especially with emphysema, upon which an acute attack has supervened dyspnœa is always considerable, and is liable to become extremely urgent, with early and grave cyanotic signs, particularly if the heart is also affected. Œdema of the extremities, or even of the trunk, readily occurs. Pain is frequently absent, but the cough is distressing and severe. In cases of emphysema, the expectoration is at first very frothy, as well as abundant. Even a slight amount of acute Bronchitis superadded to extensive chronic catarrh with emphysema, brings with it much danger.

6. MECHANICAL BRONCHITIS.—The various irritating substances such as charcoal, &c., when inspired, at first give rise to slight but repeated attacks of acute catarrh, without coryza, not attended with pain or fever, but having an exceedingly irritable and frequent cough without much expectoration, which contains some of the particles

inhaled. The condition soon becomes chronic, and will call for a few further remarks when Chronic Bronchitis is treated of.

Under this head it will be necessary to notice briefly those cases in which bronchitic symptoms are brought on by the inhalation of certain vegetable matters, the most important being " hay-asthma," or " hay-fever." The symptoms of bronchial irritation are prominent. There are frequent and severe paroxysms of coughing, but there is generally no expectoration, or at most a small quantity of clear, thin, watery mucus. Breathing is much oppressed, and there is often considerable soreness behind the sternum. Marked coryza occurs, and other indications that the whole tract of the respiratory mucous membrane is involved ; much general languor and want of energy is experienced, but fever is absent. Only a few, possessing a special idiosyncrasy, are liable to this complaint, and they are attacked on the slightest exposure to the exciting cause, and sometimes apparently even without this ; hence they usually suffer every hay season. The symptoms come on suddenly, and are severe almost from the outset ; they may last from two to six weeks or more.

Ipecacuanha produces very similar effects, and I am acquainted with a case which recently occurred, in which a severe attack resulted from smelling for a moment a bottle containing ipecacuanha powder, as an experiment, the patient having previously suffered in a similar way.

Physical Signs :—1. *Inspection.* (*a*) *Form and size of chest* rarely altered, but if the lungs are greatly distended, the chest may be somewhat enlarged, but equally so throughout. (*b*) *Movements* more frequent and more rapid than in health, in proportion to the amount of dyspnœa. Expiration is evidently difficult and ineffectual, and hence protracted. In most cases the abdominal movements are in excess of the thoracic, but if there is extensive accumulation in the tubes, the upper costal movements become considerably the more marked, and elevation is often in excess of expansion. Much, however, will depend on age, sex, the extent of the tubes involved, &c. In children, particularly if they are the subjects of rickets, signs of more or less imperfect inspiration are commonly observed. The epigastrium, ensiform cartilage, and contiguous rib cartilages sink in during each inspiratory act, the lower ribs are drawn in laterally, and the supra-clavicular regions become deeply hollow. Niemeyer mentions another sign of the same condition, viz. " prominence of the supra- and infra-clavicular regions, with feeble respiratory movements."

2. *Palpation.*—In addition to the signs mentioned under " Inspection," palpation reveals usually " rhonchal fremitus," of variable quality and extent. It may be felt over a large area, without a large number of tubes being necessarily involved ; but should it continue thus for some days, it indicates widely-spread Bronchitis. The presence of this fremitus shows that some of the more superficial tubes are affected. It generally accompanies both inspiration and expiration, but it is often more marked during one or other act. A cough

may cause it to disappear, or alter its position. Stokes states that it is more marked in females, and over the lower and middle part of the chest. It may be felt only in front, and over the upper part of the chest. This sign is of great importance in the physical examination of very young infants. Vocal fremitus varies widely, and cannot be relied on. Tussive fremitus is often well marked.

3. *Percussion.*—In most cases the area and amount of pulmonary resonance are not obviously altered. It not unfrequently happens, however, especially in children, that owing to the air-vesicles and small tubes being permanently distended in consequence of obstruction, the resonance is in excess, both in extent and degree, and is not diminished after expiration in the normal proportion. Rarely, a certain amount of deficiency in tone may be noticed over the base of the lungs posteriorly, owing to great accumulation of secretion, congestion with œdema, or lobular collapse; and the same may be observed in other parts of the chest, if collapse has resulted from obstruction of a large tube, or even extensively should the main bronchus be pressed upon by enlarged glands, which is said to happen sometimes. In infants, a sound resembling the "*bruit de pot fêlé*" may often be produced by sharp percussion, especially during expiration, variable in its site.

4. *Auscultation.* (*a*) *Respiratory Sounds.*—These vary considerably in different parts of the chest. Where the tubes are free the sounds are loud and exaggerated, and this is usually the case towards the upper part of the thorax. Over the affected regions they are weak, and may become totally suppressed, owing to the narrowing or complete closure of the tubes by thickened membrane and secretion; or temporarily, from spasm of the muscular fibres. Their quality is always harsh and coarse, and expiration is prolonged. In the early stage the sounds seem dry, but later on certain rhonchi are mingled with them, by which they may be completely masked.

(*b*) *Adventitious Sounds.*—These include the various "rhonchi" produced by the air passing through tubes containing fluid, or diminished in calibre by thickened mucous membrane or spasm. They vary with the nature and quantity of the fluids, the size of the tubes in which they are originated, &c., and are divided into "dry" and "moist." The former comprise the "*sonorous*," which are very low-pitched and grave in tone, resembling the sound of snoring generally, but sometimes of rubbing or other quality, often heard extensively, and giving an impression of superficialness in their origin; and the "*sibilant*," which are high-pitched, and may be musical, hissing, or whistling. If the sibilant rhonchi are extensively heard, it indicates that the smaller tubes are affected. Occasionally "clicking" sounds of dry character are observed. The "moist" rhonchi are all more or less bubbling, being caused by the passage of air through fluid. They vary much in size, quality, and pitch, according to the quantity and consistence of the fluid and the dimensions of the tubes in which they are produced, and the varieties are named "mucous," "sub-

mucous," "sub-crepitant," &c. Occasionally they have a "rattling" character.

It will be readily understood that these rhonchi are variously combined, and are heard in different parts of the chest, according to the seat, extent, and stage of the Bronchitis. Generally they exist on both sides, though not to the same degree, but may be localized to a part of one lung. At first the "dry" may alone be present, but the "moist" are soon added, and frequently both forms are perceptible from the first. The "moist" are usually most marked behind and towards the base of the lungs. All kinds are liable to change their sites, as well as to disappear for a time, sometimes suddenly, either from the secretions having been driven out of the tubes, or because these have become thoroughly blocked up. A strong cough will often disperse many of them. These remarks are especially true with regard to the sonorous and sibilant rhonchi.

When Capillary Bronchitis is present, abundant and very minute bubbling rhonchi are heard towards the lower part of both lungs, accompanying inspiration and expiration, and completely hiding the breath-sounds; while higher up they are larger, and the respiratory sounds are perceived, altered in quality. This may be partly the result of gravitation, but very extensive and minute rhonchi indicate that the smaller tubes are themselves implicated.

(c) The action of the heart sometimes causes rhonchal sounds.

Vocal resonance is not materially altered in either direction. The cough is generally very loud, and gives rise to a number of rhonchal sounds.

5. *Position of Organs.*—As a rule this is normal, but if the lungs are greatly distended the diaphragm is depressed, and with it the liver and spleen somewhat. The heart is said to be pushed downwards and to the right. In some cases which have recently fallen under my notice in the post-mortem room, the heart was so placed that its right border lay almost horizontally on the diaphragm, and its apex was outside the left nipple-line, occupying a similar position to that described by Niemeyer as occurring in emphysema.

DURATION AND TERMINATIONS.—In the milder forms the duration varies from four or five days to three weeks or more, but a case is usually convalescent under nine to twelve days. In fatal cases of Capillary Bronchitis death generally occurs in a few days, but it is difficult to lay down any certain average. Walshe gives from the sixth to the eighth day for children, from the tenth to the twelfth for adults. Convalescence is not thoroughly established for some time in cases that recover, but generally begins under three weeks. The clinical terminations are : (a) complete recovery, (b) death, (c) transition into the chronic state. Relapse may occur, or an extension of the Bronchitis; but this is not common. As already mentioned, it is an affection very liable to recur. It should be mentioned that it may leave behind it permanent emphysema, or may be the foundation

are the various sensations behind the sternu[m]
of oppression, often amounting to dyspnœa, w[hile]
expectoration having the characters already [of]
catarrh, as well as the general symptoms, [with]
rigors, and absence, or comparatively small [is]
important. The more significant physical s[igns]
dulness, or any material alteration in the voca[l]
the characters of the breath-sounds, but es[pecially]
the various rhonchi, as indicated by palpati[on]
the majority of cas[e] s there is no difficulty [in]
diagnosis, but doubt may arise in some instan[ces]
to notice briefly the special diagnosis of Bron[chitis]
affections.

It cannot be decided in the earlier stage of
the case is not one of Bronchitis. Subse[quently]
nature of the attacks—with the peculiar cough
followed by vomiting—is sufficiently charact[eristic]
However, it may be complicated with Br[onchitis]
revealed by its physical signs.

In some children the breathing of Bronchit[is]
resemble that of croup, the cough being at
ringing or husky, and the voice affected. The
in such a case are, the presence of catarrh;
and not truly stridulous, but wheezing; feve[r]
cough is soon moist, and expectoration may
the base of the tongue, which does not cont[ain]
brane. Physical examination also shows the

Laryngitis in the adult is distinguished b[y]
toms, which are localised in this part, and by
symptoms and physical signs of Bronchitis.

Pneumonia occurring in the adult is usual[ly]

nd vocal resonance is not of a metallic and sniffling character. In such
case attention must also be paid to the symptoms. From lobular
neumonia occurring in children the diagnosis is often difficult. In
iis affection frequently no dulness can be observed, or it may be
resent in Bronchitis from collapse. In the latter the moist rhonchi
re much more diffused, and of larger size; at first they are generally
nited to, and throughout are most marked at the bases, whereas in
bular pneumonia they are scattered irregularly. Tubular breathing
not heard in Bronchitis. There is less fever, and the skin is not
idly hot, being often moist. The respirations are more frequent in
ular pneumonia, but the sense of dyspnœa is much less, as well as
e asphyxial appearances and general anxiety.

The symptoms and physical signs of pleurisy are so totally dif-
ent from those of Bronchitis, that it does not appear necessary to
anything as to their diagnosis.

When a child is attacked with bronchial symptoms, it is sometimes
ficult to determine at first whether they constitute the entire
ment, or are associated with one of the exanthemata. The amount
fever as evidenced by the thermometer, and the special symptoms
emonitory of the various fevers, must be the guides until the erup-
n appears. The same applies to typhoid fever, at any age, which
iy be at the early period masked by the bronchial catarrh if its
iinary symptoms are not prominent. The thermometer will prove
great value in any such cases.

From the various forms of acute phthisis, Capillary Bronchitis may
distinguished by the following characters. The fever is much less,
d consequently the temperature is considerably lower; the pulse-
spiration ratio is less perverted; signs of asphyxia set in; there is
e expectoration of a muco-purulent character. There are abundant
and moist rhonchi, the latter being most marked below. In one
m of acute phthisis there are signs of consolidation, followed by
ose of cavities. In the true tubercular miliary form there are
rdly any physical signs except scattered rhonchi, which are most
ındant towards the upper part of the lung. The dyspnœa is very
at, and there is violent fever.

PROGNOSIS AND MORTALITY.—As will be seen from the tables given
en considering the causes of Bronchitis, this is a disease attended
th much danger, especially if it be extensive. Its prognosis must
guided by the following circumstances:—1. *Age.* The mortality is far
eater among children, especially young infants, and the aged, than in
ults. 2. *State of health.* The danger will be increased in propor-
n as this is below par, and particularly if there is any positive
sease present, either acute or chronic. 3. *Extent of inflammation.*
both lungs are widely affected with Capillary Bronchitis, the
ognosis is grave, even in healthy adults. 4. *Previous state of the
igs and heart.* Any chronic disease of these organs will seriously
gravate the danger, but especially extensive emphysema, with dila-

emphysema; pleurisy; gastric or intestinal catarrh
character. 8. *Time and method of treatment.* The soo
care and treatment are adopted, the more likely i
brought to a favourable issue.

PATHOLOGY.—Bronchitis is in most cases a catarrha
of the mucous membrane lining the bronchial tube
associated with a similar condition in the trachea.
becomes hyperæmic, and, as a result of this, excessiv
poured out into the tubes, as well as into the substance
Nutrition is perverted, and an excessive formation of c
on the surface of the membrane; these are thrown of
less imperfect state, and, mingling with the fluid, g
various corpuscles seen in the expectoration, to which
owes its increasing opacity. In many cases it is a pu
plaint, the result of direct irritation; in others it is
some general condition of the system, produced under
"cold" and other agencies, in which the mucous meml
liable to suffer more or less extensively. Again, in
it appears to be an attempt on the part of the
assist in throwing off some morbid material containe
which is attended with congestion. With regard to
"Capillary Bronchitis," in many instances undoubted
properly applied. There is an actual inflammatory stat
tubes, which may either extend from the larger tub
there in the first instance, or, I believe, may in some c
by the irritation of secretions formed in the larger
back into the smaller. But in other instances there a
of any inflammation in the capillary tubes, and it s
that there is merely a collection of fluid in these tu
flowed down from those of larger calibre, in consequen
power to expectorate. This would be aided by gravita
by the destruction of the ciliated epithelium, and, aft

is a part of the general state. None is present if the affection is local and limited. The asphyxial symptoms are easily explained by the obstruction in the air-tubes, and consequent interference with the due aëration of the blood. In proportion as the vital powers are below par, will be the tendency of the combined fever and imperfect respiratory process to lead to a fatal result.

MORBID ANATOMY.—On opening the thorax of a person who has died from extensive Bronchitis, the lungs do not collapse, but remain distended, or may even bulge out: this is caused by the air being unable to escape through the obstructed tubes, and even pressure cannot materially diminish their bulk. The degree of this distension will, of course, vary with the number and size of the tubes affected. The mucous membrane presents various forms of redness, and generally all are seen more or less in the same case. Thus, it may be arborescent, capilliform, mottled, streaked, in points, or diffused, but it is not uniformly spread over a large surface as a rule. In tint it may vary from a bright, vivid pink-red, to a somewhat dark, venous hue, the latter being observed in the later stages. It is sometimes scarlet, and has a velvety appearance. The redness does not ordinarily extend beyond the fourth or fifth divisions, often not beyond the second or third, but it is said that even the finest ramifications may exhibit it. It is generally more marked towards the upper part of the lung. Possibly the action of the elastic and muscular fibres in the walls of the bronchi may diminish it after death. Where the tubes bifurcate, it is often well marked. Thickening and opacity of the membrane are also observed, to a greater or less degree, from distension of the vessels and infiltration into its substance; from this cause, as well as frequently from the presence of exudation in the sub-mucous tissue, the tubes are reduced in calibre, but unequally, and the surface of the membrane appears uneven. The more minute tubes may be completely closed up; and this is especially apt to happen in young children. The tissue of the membrane is relaxed and softened; often it cannot be stripped off for any length. Patchy abrasions of the epithelium are frequent, giving sometimes an appearance of slight ulceration, but this is never observed in children (Vogel). In the very early stage abnormal dryness is observed, or a very small quantity of transparent tenacious substance covers the surface. Soon excessive secretion is formed, and various materials are found in the tubes, corresponding to the different stages of the expectoration. They may be so abundant as to extend from the finest ramifications up even to the trachea, completely filling all the canals. In appearance the contents of the tubes resemble frothy mucus, or a muco-purulent, or even purulent-looking fluid; the degree of viscidity and adhesion to the surface of the membrane varies, but is usually marked. More or less blood may be present. Sometimes a fibrinous-looking material is seen attached to the surface, lying loose in flakes or masses, or even forming complete

casts of the smaller tubes, which may be hollow or solid. The microscope reveals epithelium scales, perfect and ciliated in the early stage, but afterwards imperfect, small and somewhat oval in shape; so-called mucus and exudation corpuscles; large pus-corpuscles, containing numerous granules; sometimes blood discs; granular material in abundance. More cells are observed in proportion to the opacity of the fluid. It is in the lower and more dependent parts of the lungs that the secretions are found in largest quantity. In some cases, especially in children, small yellow spots are visible near the surface, due to accumulation in the air-cells and minute tubes.

Along with the Bronchitis, and as the result of it, lobular collapse is very commonly observed, as was first pointed out by Dr. Gairdner. This condition is particularly frequent in young children. If a large tube is blocked up, more extensive collapse is present. In some cases the bronchial tubes are slightly but uniformly dilated, and acute emphysema is said to occur, but it is a question whether in these cases the air-vesicles are usually actually distended beyond their normal size in deep inspiration, and therefore whether the term emphysema can be properly applied to this condition. Lobular pneumonia is occasionally present, and may be preceded by collapse. Ordinary lobar pneumonia is rare. There may be more or less congestion of the lungs with œdema, or these organs may be natural in hue, or even paler than normal. The bronchial glands are often large, red, and softened.

The blood is dark, and the venous system, with the right side of the heart, overloaded.

Of course the morbid appearances characteristic of Bronchitis will vary according to its extent. Both lungs are usually involved, but seldom to the same degree; nor is one lung affected uniformly throughout. Different conditions are seen in different parts of the same lung. The membrane lining the smaller tubes suffers less than that of the larger, but more secretion is contained in the former. In cases of death from other causes, more or less Bronchitis is often present.

TREATMENT.—No case of Bronchitis, however slight, should be neglected, because a little care and appropriate treatment at the outset may soon put an end to an attack which otherwise might become very serious or even lead to a fatal result. It must be remembered also that a catarrh, if overlooked at first, may lay the foundation for certain incurable chronic affections. The treatment will necessarily vary much according to a variety of circumstances, and therefore no uniform method can be laid down. I shall first consider the mode of dealing with an ordinary case resulting from cold, and afterwards notice any modifying conditions which may appear to call for remark.

It is always well, if possible, to make the patient keep to the house, or even to one room, maintained at a uniform temperature of

64 to 66° Fahr., if the weather is at all unfavourable ; but under any circumstances damp night air must be avoided. Lying up thus for two or three days will often cure a catarrh. It is customary as well as, I believe, useful in these cases, to endeavour to bring about a free action of the skin. For this purpose a copious warm drink may be given before going to bed, such as hot milk, mulled claret, warm elder wine, or even some strong alcoholic stimulant, such as hot spirit and water. A warm foot-bath should be used, and some mustard and salt may be added to the water. A large quantity of bed-clothes should be put on the bed, and the patient should sleep between blankets. Finally a full dose of Dover's powder may be administered, or a diaphoretic and saline draught. Some recommend a hot-air or vapour bath, and a Turkish bath has certainly often the effect of curing a cold. Wrapping the body in wet sheets is employed by some in order to procure free perspiration, and does not seem to be attended with danger. It is generally advisable to apply a large mustard poultice over the front of the chest, and to allow steam to be inhaled for a few minutes.

If the case is a severe one from the first, and attended with fever, or if it is not checked by the above treatment, more active measures will be required.

Venesection has been practised extensively in this disease. In most cases it certainly is not required, while in those of a more serious type it is extremely rare to meet with the combination of conditions which warrant the taking of blood from the arm. These conditions are said to be where the inflammation is marked and extensive, occurring in a robust and healthy young or middle-aged adult, and accompanied with severe sthenic fever. It can be safely affirmed that venesection is scarcely ever called for, at all events in town districts. *Local bleeding* by leeches or cupping may certainly be employed with advantage in some cases, but great discrimination is necessary even in the use of these modes of removing blood, and in the great majority of instances they can well be dispensed with.

If leeches are used, they should be applied over the front of the chest, or sometimes at the base posteriorly. Their number must vary according to circumstances, but certainly more than from five to ten are seldom advisable. In plethoric children, the blood removed by two or three leeches sometimes relieves great dyspnœa very effectually. Cupping may be performed either in front or behind, if thought necessary, to the extent of from three to six ounces.

It is certainly improper to adopt, as the ordinary practice, any mode of removing blood in cases of Bronchitis; it is far safer to act on another principle. In any doubtful case the patient will stand a better chance of recovery if no blood is taken away. Free dry cupping over the chest, both front and back, is often of much service, relieving the oppression and dyspnœa, and it is quite devoid of danger.

An *Emetic*, in the form of tartar emetic, or ipecacuanha, is made use of by some at the outset, especially in children. Though ex-

tremely valuable in certain conditions, it appears to me that it may well be dispensed with at this time. The bowels may be freely opened by some aperient, varying according to the age and condition of the patient, and throughout the case mild purgatives must be used as required.

Among medicinal substances, *tartar emetic* ranks as one of the most important during the early stage of Bronchitis. The dose must be regulated by circumstances, but from a third to half a grain every four hours is usually sufficient for an adult. It may be given in a saline draught, containing liquor ammoniæ acetatis, and its effects should be carefully watched.

Tincture of digitalis is also employed by some at this period of the case, and often with marked benefit. *Calomel* with *opium* is recommended if either of the above cannot be taken from any cause, but it seems to me of very doubtful efficacy, and might be often injurious. As the case progresses, and secretions form in the bronchial tubes, the main indications which medicines have to fulfil are the following:— 1. To assist expectoration. 2. To alleviate cough, due regard being had to the proper discharge of the secretions. 3. To diminish the quantity of the expectoration. 4. To allay spasm of the tubes, if present. These are carried out by the administration of various expectorants, sedatives, narcotics, and antispasmodics, in different combinations, along with diaphoretics or demulcents. The chief expectorants are ipecacuanha wine, tincture or oxymel of squills, compound tincture of camphor, and, later on, sesquicarbonate of ammonia or chloride of ammonium, senega, serpentary, galbanum, ammoniacum, tincture of benzoin and balsam copaiba. The sedatives and narcotics principally used are hyoscyamus, conium, opium or morphia, and hydrocyanic acid. The most important antispasmodics include sulphuric ether, æthereal tincture of lobelia, and spirits of chloroform. These various medicines must be employed as they are required, and no rules can be laid down as to their precise use; but it may be stated that the less stimulating expectorants should be given at first, and narcotics, especially opium, must be used with very great caution if expectoration is difficult, and the secretions tend to accumulate.

Local Applications.—Sinapisms are beneficial even from the first, and may be repeated over different parts of the chest. Hot applications are also of much value in the early period, especially hot moist flannels, which may be sprinkled with turpentine, or linseed-meal poultices, which should be large, applied very hot, changed frequently, and continued for some time. The latter are particularly valuable in children. Blisters are called for after expectoration sets in and the acute symptoms have subsided. One of good size may be placed over the front of the chest, or some recommend the interscapular region behind as the best place for a blister, because more fluid will be drawn there, but the discomfort caused is a great objection. In children the blister may be left on only for two or three hours, and afterwards a linseed-meal poultice applied.

If the affection is tending to become chronic, other forms of counter-irritation may be employed, as the application of croton oil liniment, or acetic acid.

Inhalations.—In the early stage inhalations of simple steam are decidedly useful in many cases, especially if the larynx is in an irritable condition, and giving rise to constant cough. Later on medicated inhalations are of service under certain circumstances, such as those of conium, sulphuric ether, or chloroform, if there is much spasm; those of tar, or creosote, if the expectoration continues very abundant.

Diet and Regimen.—It is quite unnecessary to keep patients on too low a diet, and they may have a fair quantity of beef-tea and milk from the first. With regard to stimulants, it is impossible to lay down any positive rules. Ordinarily they are not required, and might be injurious; but if there are any indications for their administration, such as a tendency to adynamia or asphyxia, they should be given without delay, and their effects watched. Any case that is at all severe should be absolutely confined to the bedroom, kept at a temperature of 66° to 68° Fahr. the air being moistened by steam from a kettle kept constantly boiling; at the same time the room should be occasionally well ventilated, the patient being protected from draughts. Warm clothing must be worn, including flannel, with a sufficient amount of bed-clothes. The head should be kept high, and cough should be encouraged, if there appear to be any indication that the secretions are not properly discharged; on the other hand, an irritable cough may sometimes be subdued by an effort of the will.

In treating children, emetics are useful at the outset, if the attack is severe. For ordinary cases, ipecacuanha wine constitutes a valuable medicine. They should be encouraged to cough, if old enough, and means should be used to make them breathe freely and expectorate; sleep must not be too prolonged or deep. The throat should be cleared occasionally from mucus by means of the finger; and if the physical signs show that fluids are accumulating in the tubes, an emetic ought to be given, the best being sulphate of zinc. Narcotics must be used very cautiously in children. They should drink freely. Attention must be paid to their constitutional state, such as rickets, tuberculosis; and if either of these exist, all depressing treatment must be carefully avoided. The quality and quantity of the milk should be looked to.

In aged persons and those who are debilitated or are suffering from any acute or chronic disease, depletion in any form is inadmissible. Antimony can only be given very cautiously, and, in most cases, stimulants and stimulant-expectorants are required from the first. Wine or brandy, with plenty of strong beef-tea and other nourishment, must be administered, in quantities according to the requirements of the case. Sesquicarbonate or muriate of ammonia, cinchona, camphor, sulphuric ether, spirits of chloroform, senega, squill, galbanum, ammoniacum, are the medicines called for.

In most cases of Capillary Bronchitis the stimulant treatment is decidedly that which yields most favourable results. If the Bronchitis originates from any constitutional condition, such as gout or tuberculosis, the treatment appropriate for these affections must be employed.

When an acute attack supervenes upon Chronic Bronchitis, free dry cupping, with flying blisters over the chest, are very serviceable. If asphyxial symptoms set in, the stimulant treatment must be persevered in. Chlorate of potash may be given frequently as a drink; artificial respiration and galvanism along the course of the vagus nerve are recommended in extreme cases, and the former may be carried out in children persistently whenever there are signs of danger. A warm bath, with cold affusion while in it, is also advocated by some as an effectual mode of combating asphyxia. If there is a great amount of fever, and there seems to be danger from this cause, quinine is advisable, in doses of from two to three grains every three or four hours.

During convalescence, tonics, such as quinine, mineral acids, iron, may be added to the other medicines. The clothing should be warm, and a pitch-plaster may be worn on the chest. Cold and damp must be avoided for some time, and the patient should not neglect proper precautions until perfectly convalescent.

In those who are subject to Bronchitis, prophylactic measures are called for. All causes likely to bring it on must be avoided; if possible, a change to a warm climate during the cold season should be enjoined. Cold sponging is useful, especially for children, who should be properly clothed, but not immoderately. Constitutional treatment may be called for.

With regard to the treatment of hay-asthma, during the attack small doses of dilute hydrocyanic acid, with tincture of lobelia and other antispasmodics, frequently repeated, seem to act best. Inhalations of creosote are also recommended, or of an atmosphere containing a small proportion of chlorine. In the intervals general tonic measures are useful, such as quinine and iron, with cold bathing. Arsenic and nux-vomica are also employed. Dr. Reynolds has found much benefit from the systematic inhalation of chloroform, from a handkerchief, four or six times a day.[1] In the spring any one subject to this affection should go to the sea-side or take a voyage.

CHRONIC BRONCHITIS. CHRONIC BRONCHIAL CATARRH.

CAUSES.—Much that has been said on this head with regard to Acute Bronchitis applies to the chronic form also. In fact, it usually is the result of the acute affection, remaining sometimes even after a single attack, especially if the deeper structures of the tubes have become involved, but in the great majority of cases being due to

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[1] See *Lancet*, "On the Value of Chloroform Inhalation in certain Classes of Spasm."

several repeated attacks. The complaint may be chronic from the first, coming on slowly; and this is particularly the case in old people. It is commonly associated with any chronic lung disease which may exist, but especially emphysema or dilated bronchi and the various forms of phthisis. In the latter affection much of the expectoration arises from a chronic catarrh of the mucous membrane. The presence of certain heart diseases, interfering with the circulation, frequently leads to Chronic Bronchitis, especially some forms of it. Various blood affections, notably gout, predispose to it greatly. The inhalation of the different irritating particles, already alluded to, soon cause the affection to become permanent and chronic. Old people suffer in a much larger proportion than those who are younger, but the complaint may be present even in children, particularly after measles and hooping-cough, or where tuberculosis exists. It frequently exists in connexion with chronic alcoholism.

SYMPTOMATOLOGY.—The local symptoms of Chronic Bronchitis are in kind similar to those attending the acute form, viz. cough, usually accompanied with expectoration, more or less oppression in the chest or dyspnœa, and frequently uneasiness behind the sternum, which, however, is never considerable, and may be absent. These differ materially as to their degree in different cases, and they are variously combined, while certain conditions of the lungs and heart modify them, so that it becomes necessary to classify them into certain groups. The constitution may not be affected in the least, or, on the other hand, may suffer severely.

It will be necessary to describe three chief forms, which are mainly distinguished by the quantity and characters of the expectoration attending each variety, and the qualities of the cough.

1. The first group about to be considered includes the ordinary cases of Chronic Bronchitis, which present innumerable grades of severity, both in the intensity of the symptoms and in their degree of persistence, and the same case will often exhibit these grades during its progress.

At first the patient is only attacked during the winter, having what is termed "winter cough," and being perfectly well during the warmer months. Afterwards the attacks become more frequent, until finally the complaint becomes permanent, but is always aggravated whenever cold weather sets in. The cough may not be severe, only occurring in comparatively slight paroxysms, at considerable intervals; or it is more or less constant, but also increased in paroxysms, which are often very violent and distressing. They are worse at night usually, on first going to bed, but they are especially severe in the mornings, on account of the secretions having accumulated. In bad cases, sleep is much disturbed by fits of coughing during the night.

The expectoration may be brought up without much difficulty in the less advanced cases, but later on it is discharged only with great trouble, as well on account of its own characters as the state of the

bronchial tubes, in the smaller of which it is chiefly formed. It varies in quantity greatly, being sometimes inconsiderable, at other times exceedingly abundant. In the slighter cases, it consists of yellowish-white or greyish masses of muco-purulent matter chiefly, containing a number of young, imperfect cells. In others it may be partly in the form of viscid, tenacious, greyish mucus, but the greater portion of it is muco-purulent or purulent in appearance, usually of a yellowish white colour, but often greenish-yellow, and sometimes bright or dark green. As a rule, it is not much aërated, and often not at all; hence it may sink in water, either partly or entirely, but often floats. The different masses may remain distinct, but usually run more or less together. True "nummulated" masses of large size are occasionally seen. All the forms of expectoration tend towards opacity, and it is not uncommonly thoroughly opaque; an exceedingly disagreeable odour is often present in bad cases, which may amount to extreme fœtor. This is supposed to be due to some chemical change, and butyric and other acids have been detected, as in a case of Dr. Laycock's. Minute microscopic sloughs of the mucous membrane have also been considered as its cause. Streaks of blood may be observed, especially if the cough is very violent and expectoration difficult, and still more if heart-disease exists. With the aid of the microscope, abundant imperfect epithelium-cells are seen, with pus-cells and granular matter. Blood-corpuscles are frequently visible also.

There may be not the least uneasiness or pain behind the sternum, or it may be present at first, and afterwards cease. Generally a certain amount exists, and particularly a sense of soreness after severe cough. In severe cases, breathing is somewhat short on exertion, and during the fits of coughing the respirations are increased in number, but evident dyspnœa does not exist, unless there is emphysema.

The constitution does not suffer in the milder attacks, and the general condition is unimpaired; but in permanent and extensive Chronic Bronchitis the system is gravely affected, on account of the interference with sleep, abundant expectoration, and other circumstances. The flesh wastes, and emaciation may become marked, but it does not go beyond a certain point, and then remains stationary. The strength is reduced in proportion to the wasting. A slight degree of fever often sets in towards the evening, followed by copious sweats at early morning, and there may be marked hectic fever. This increases the loss of flesh materially. The digestive organs usually suffer to a greater or less extent, as evidenced by a furred tongue, deficient appetite, and constipation. If the system is much implicated, the patient is quite unable to follow any employment.

2. A very characteristic class of cases is that which is described under the terms "dry catarrh" ("*catarrh sec*" of Laennec), or "dry bronchial irritation." In this form of Bronchitis very little secretion is produced, and that principally in the smaller tubes.

The mucous membrane is in an exceedingly irritable state, and hence violent, prolonged, and very distressing fits of coughing come on, during which the face becomes turgid and red, and the veins swell out, the smaller vessels at last remaining permanently distended. There is, as a rule, no expectoration at the close of the paroxysm, but sometimes a small mass of tough, viscid, semi-transparent greyish mucus is discharged, compared to boiled starch or pearl, or a little thin watery fluid. Much soreness is frequently experienced in the chest after a spell of coughing. There is persistent shortness of breath, which may amount to extreme dyspnœa at times, should a large bronchus be blocked up, or acute catarrh set in. In some instances spasm of the bronchial tubes evidently exists. A feeling of constriction about the chest is always present. Vomiting may occur during a paroxysm of cough. Febrile symptoms are usually entirely absent, but there may be an occasional slight rise of temperature; as a rule the general condition is unaffected. This form of Bronchitis is liable to lead to emphysema, and is commonly associated with this condition in a variable degree. It is frequently met with in gouty people, and is said to be prevalent at seaside places, and to come on after the cure of chronic cutaneous diseases, and in those weakened by excesses.

3. The third variety is named " bronchorrhœa," which, as its name indicates, is distinguished chiefly by the abundance of the expectoration, but also partly by its characters. It often occurs in old, feeble persons, after several attacks of acute Bronchitis, particularly when there is some heart affection present obstructing the circulation. The cough comes on in paroxysms, which are often spasmodic and severe, but may be slight compared with the quantity of expectoration. A fit may set in every day, or even several times a day, and it ends with, and is relieved by, profuse expectoration, which is almost clear, transparent, thin and watery; or thick, ropy, and glutinous, compared to unboiled white of egg mixed with water. It is a little frothy on the surface, but the general mass contains no air. The quantity discharged may be very great, sometimes amounting to four or five pints in the twenty-four hours, and frequently a quarter or half a pint is expelled within an hour; and the amount of fluid poured out into the tubes may be so excessive as to cause fatal exhaustion or asphyxia, especially in aged individuals who are unable to expectorate. During the paroxysms there is considerable dyspnœa, but at other times this is not much observed. The strength fails and the flesh wastes in severe cases, but the constitution may not suffer for years if the expectoration is limited, and it may even relieve the local symptoms produced by certain forms of cardiac disease, which lead to congestion and inflammation of the lungs.

With regard to the form of disease produced by irritant particles, all that it seems necessary to add here is, that after a while the symptoms of bronchitic irritation become chronic, with a constant dryish cough, and subsequently consolidation takes place, which

leads to destruction of the pulmonary tissue, and thus cavities are ultimately produced. At first there are no general symptoms, but afterwards emaciation and exhaustion set in. Usually the course of these cases is very chronic, but it may be tolerably rapid.

It must be borne in mind that very rarely does Chronic Bronchitis exist in an uncomplicated form, and its symptoms will be materially modified by co-existing states of the lungs and heart, and also by the constitutional condition of the patient.

Physical Signs.—It is difficult precisely to define what physical signs are associated with Chronic Bronchitis as its direct results, because there are so many other morbid conditions generally added to it.

1. *Inspection.*—In ordinary cases there is nothing abnormal in the form and size of the chest, but it may be equally and generally enlarged, especially in dry catarrh, in which it is drawn up and made more convex; but here, probably, more or less emphysema exists. The movements are deficient in bad cases, especially that of expansion, and expiration is seen to be prolonged and laboured.

2. *Palpation* reveals rhonchal fremitus over various parts of the chest, subject to changes in amount and site. The vocal fremitus is not obviously altered.

3. *Percussion.*—In most instances of confirmed Chronic Bronchitis, it will be found that the resonance is increased in extent and degree on account of co-existing emphysema. Similar conditions to those mentioned under the acute form may cause temporary and localized dulness.

4. *Auscultation.*—The breath-sounds are much weakened usually, but vary in different parts of the chest. After a free expectoration they may be heard extensively. Their quality is always harsh and coarse, and sometimes markedly so. In unaffected parts they are exaggerated. Expiration-sound is much prolonged in long-established cases. Rhonchi of various kinds are heard, but the "dry" are most abundant. The "bubbling" rhonchi are of large, coarse character, and are often temporarily absent. They are altered by cough and deep inspiration, as in the acute affection. It will be readily understood that these rhonchi will vary in the different kinds of Chronic Bronchitis, according to the quantity and consistence of the fluids contained in the tubes. In bronchorrhœa the bubbles give the idea of being produced in a thinner fluid than that of the ordinary form, while in dry catarrh they are necessarily absent. Vocal resonance is very variable. It may be bronchophonic, normal, deficient, or absent.

5. *Displacement of Organs.*—Owing to the emphysema accompanying Bronchitis, there is usually more or less displacement, particularly of the heart.

DIAGNOSIS.—There is scarcely ever any difficulty in diagnosing the presence of Chronic Bronchitis, but this is frequently experienced in determining with what conditions it is associated. Where there is much emaciation, with abundant purulent expectoration, it may

simulate certain forms of phthisis, but the comparative degree and rapidity of wasting, absence of or only slight fever as a rule, want of hæmoptysis, and other symptoms present in phthisis, as well as the physical signs, will distinguish them. It is only when there are dilated bronchi, that usually much difficulty is felt in arriving at a correct diagnosis, and these cases are considered elsewhere.

PROGNOSIS.—No case of Chronic Bronchitis ought to be looked upon as unimportant or treated lightly. Though it does not of itself often cause death, yet in proportion to its extent does it become dangerous, as an acute attack may set in at any moment; and however slight this may be, the danger in such a case becomes considerable, on account of the difficulty in expelling the secretions from the tubes. At the same time it is a serious affection, because it tends to become more diffused, and also to give rise to certain important and incurable sequelæ. These are chiefly emphysema, dilated bronchi, and collapse. Many pathologists believe, also, that by extending into the air-vesicles it may be the immediate cause of a form of phthisis; in fact, what they consider the ordinary form of pulmonary consumption. Others are of opinion that by causing irritation it leads to a deposit of true tubercle. Much will depend upon the variety of the disease which is present, the amount of expectoration, its effect upon the constitution, the age of the patient, the state of the lungs and heart, and other circumstances. It is said that complete and permanent recovery may take place in the young, if they are taken to a proper climate and treated carefully. In almost all cases it is incurable, when once it is well established.

PATHOLOGY AND MORBID ANATOMY.—Many cases of Chronic Bronchitis are simply due to congestion, usually passive, sometimes more or less active, of the bronchial mucous membrane; others present a so-called inflammatory condition of the membrane, also involving the deeper structures after a while. Hence there is permanent hyperæmia, with perverted nutrition and excessive cell-formation. The morbid appearances met with are usually as follows:—The mucous membrane is discoloured, being of a more or less dull red, often of a deeply venous hue; a dirty greyish or brownish colour may mingle with the redness. It is usually in patches, but may be diffused extensively. The minute vessels are dilated, and frequently varicose. Swelling and increased consistence of the membrane are usually marked characters; hence reduction in the calibre of the tubes, and an uneven surface. The submucous tissue in time becomes infiltrated, contracted, and indurated, thus in some parts completely closing up the smaller tubes, while the larger tubes tend to dilate diffusely, or even saccularly; a fibroid material is produced, which may increase, and at last fibroid phthisis be established. The elastic and muscular coats of the tubes become hypertrophied, but their elasticity is lost. The cartilages are prone to be the seat of calcareous deposit. Owing to the thickening

and induration of their walls, the tubes gape when cut across, and many appear enlarged. Epithelial abrasions are common and diffuse, or follicular ulceration is said to be observed occasionally, especially in tubercular phthisis. The contents of the tubes correspond to the matters expectorated. There is often a large quantity of yellowish purulent-looking fluid, which may completely fill the smaller bronchi. Usually frothy mucus exists in the larger ones. In dry catarrh the membrane is much swollen, and has upon it a small amount of tenacious, glairy, semi-transparent mucus. Emphysema is constantly present to a greater or less extent.

TREATMENT.—Much harm unquestionably results from the indiscriminate treatment of cases of Chronic Bronchitis by expectorants and narcotics, which is often practised. There is no disease in which a careful study of each individual case is more required than this, in order to take proper measures for its relief. Of course the primary object that should be kept in view is the cure of the complaint; but, failing this, it is very important to prevent it from extending, and hence early and persistent treatment is called for, not merely with the aid of medicinal agents, but also with regard to general management and hygienic measures. In advanced cases, all that can be done is to relieve certain symptoms, and to ward off various dangerous complications.

In dealing with any particular instance of the disease, the following points should be taken into consideration:—1. Whether there is any obvious cause, either external to the individual or depending upon some internal condition, which keeps up a state of congestion of the bronchial mucous membrane, and consequent catarrh. 2. The constitutional state of the patient, and the degree to which the system has become affected. 3. As regards the immediate symptoms, the treatment must depend upon (*a*) the quantity of secretion formed, and the degree of difficulty which is experienced in its discharge; (*b*) the condition of the mucous membrane; and (*c*) whether there is any spasmodic element present in connexion with the muscular fibres of the bronchial tubes.

With regard to the immediate cause of the affection, if it is known to result from any irritant inhalation, removal from exposure to this is the first thing called for. The same applies to the atmosphere of any particular district which may appear to disagree; a change to some other atmosphere is necessary, as will be pointed out when the subject of climate is considered. Certain cardiac affections seem to keep up bronchitic symptoms, by inducing congestion of the mucous membrane; when any such is present, treatment directed against it may afford much relief, especially the administration of tincture of digitalis in moderate doses, which may be combined with such other remedies as the case requires. This drug is especially recommended in the variety named "bronchorrhœa."

Various constitutional conditions are associated with Chronic Bron-

chitis, and these demand careful attention. If plethora exists, this must be reduced by appropriate diet and general management, and the use of watery purgatives. On the other hand, an anæmic state of the blood must be rectified by the different preparations of iron, which are frequently of much value. In many instances a gouty *diathesis* is present, especially when "dry catarrh" is the form of the affection assumed; if such be the case, colchicum with alkalies often proves of much service. Alkalies are also useful if there is a rheumatic tendency, as well as sulphur, certain mineral waters, and other remedies employed in rheumatism. Iodide of potassium is said to afford much relief in certain cases, and probably those accompanied with rheumatism would be most benefited. When the complaint occurs in children, in connexion with rickets or tuberculosis, the treatment requisite for these diatheses must be thoroughly and perseveringly carried out. In the great majority of cases of Chronic Bronchitis, it will be found that a general tonic plan of treatment is attended with most success. A course of quinine, or mineral acids with decoction of cinchona or some bitter infusion, often proves of great service. The quinine may be combined with sulphate of iron, or some other chalybeate preparation. Mineral nervine tonics, such as sulphate or oxide of zinc, are also of use in some cases. It is especially in those instances where there is excessive expectoration, and consequent loss of flesh and strength, that tonics are valuable; and here *also* cod-liver oil is of essential service, given in small doses at first, which may be gradually increased. Bronchorrhœa is also much benefited by tonics, especially the different preparations of iron. A course of mercury is said to have a very favourable influence over some cases of Chronic Bronchitis.

The *Symptomatic treatment* is often attended with much difficulty, and remedies have to be variously combined, and frequently changed, in order to afford relief. It will be necessary to consider briefly the main indications. The secretions may be formed in excessive quantity, and then the indication is to limit their formation. For this purpose various inhalations are of much importance. Among these, tar and creosote, or naphtha with steam, rank high. The vapour of iodine, chlorine gas, muriate of ammonia, the different balsams, and resins, are also used with success as dry inhalations. They should be employed freely diluted, and their effects carefully watched; but when properly administered they certainly often prove efficacious.

General tonic measures are called for here, and iron, especially its astringent preparations, as the tincture of the sesquichloride, is of much value. Other astringents must be given also, such as tannic or gallic acid, acetate of lead, and the mineral acids; also the various resins and balsams, especially galbanum, myrrh, ammoniacum, and balsam copaibæ; the last-mentioned is often very useful. Muriate of ammonia has been recommended. This treatment applies generally to cases of bronchorrhœa.

The fluids may not only be produced in excess, but there is also a

deficient power of expectoration, owing to the state of the tubes, the adhesive character of the secretion, or other causes. Under these circumstances stimulant expectorants are required, and may be combined with the former class of remedies. The chief of these are sesqui-carbonate of ammonia, muriate of ammonia, squill, senega, serpentary, camphor, and tincture of benzoin, in addition to the resins. Alkalies, such as the carbonate of potash or soda, or liquor potassæ with balsam copaibæ, may be tried along with ammonia, if the expectoration is very adhesive and viscid. If there is any tendency to great accumulation, an emetic of sulphate of zinc occasionally will do no harm and may give much relief. Narcotics and sedatives, but particularly opium, must be either avoided or used only with great caution in these cases, particularly in old persons ; and the patient should be encouraged to cough frequently, in order to prevent accumulation.

In other instances, the mucous membrane is in an extremely irritable state, but scarcely any secretion is produced; hence there is constant cough, with scanty or no expectoration. Should there be any sign of irritative fever under these circumstances, small doses of tartar emetic or ipecacuanha wine may be given. The most important drugs in these cases, however, are the narcotics and sedatives, which should be administered in full doses. Opium is of essential value here, and may be combined with ipecacuanha, in the form of Dover's powder, or it may be given as the tincture, Battley's solution, or compound tincture of camphor. Solution of morphia is also extremely useful. Hydro-cyanic acid, tincture of lobelia, hyoscyamus, conium, stramonium, belladonna, are other beneficial agents, and may be variously combined with other medicines. Gout is frequently present, and hence alkalies and colchicum may prove efficacious. Inhalations are to be recommended here also, but of the sedative class, viz. conium, hyoscyamus, stramonium or ether, with steam.

When there is evidently much spasm, as shown by the breathing and cough, the narcotics and sedatives are likewise employed with advantage, associated with different ethers, especially sulphuric ethers. Tincture of cannabis Indica appears to act well in some cases. Ipe-cacuanha and tartar emetic, in doses sufficient to nauseate, but not to cause vomiting, are also recommended. A few drops of chloroform may be inhaled occasionally if the tendency to spasm is great, and the sedative inhalations previously mentioned may be employed. In these cases there is always more or less emphysema. The symptomatic indications just considered are generally associated to a greater or less degree in practice, and hence the remedies have to be given in various combinations.

Local Treatment.—Free dry-cupping over the chest is often very serviceable, especially in case of irritable mucous membrane. Different forms of counter-irritation should be employed according to circumstances, viz. sinapisms, blisters over different parts, croton oil liniment, turpentine, acetic acid, or tartar emetic ointment. The croton oil liniment is certainly often very beneficial. Some recommend

an issue or seton. When these are not being used, a large warm plaster, such as a pitch-plaster covered by a thick layer of cotton-wool, should be worn over the chest in front.

Under no circumstances does it appear necessary or desirable to remove blood, either generally or locally, in cases of Chronic Bronchitis; and if an acute attack supervened, the less this mode of treatment were followed, the better would be the patient's chance of recovery. Stimulants, such as sesquicarbonate of ammonia, with chloric ether and squills, as well as wine or brandy, should decidedly be employed in preference when this happens.

General Management.—This requires careful attention. It is necessary that the patient should breathe an atmosphere of good, uniform temperature, without excessive moisture, and should avoid sudden changes. Most patients cannot leave this country during the winter season, and then they should remain indoors when the weather is at all severe, their room being maintained night and day at a regular temperature of 62 to 65° Fahr., and should always wear a respirator when out. Especially must night air and cold winds be avoided. If possible, they should reside in a part of the country possessing suitable atmospheric conditions, which vary in different cases. An entire change of climate to some more temperate region is of the greatest importance, if it can be obtained, or a long voyage may be taken. Different forms of Bronchitis require different climates; but they all require tolerably warm temperature, without sudden changes, a moderately high altitude, and protection from cold winds. For "dry catarrh" a soft and relaxing atmosphere, with moderately high temperature, is recommended. One more or less stimulating, dry and hot, is advised where there is much expectoration. In this country the principal places which receive this class of invalids are, Torquay, Penzance, Bournemouth, Grange, Clifton, and Tunbridge Wells. Among foreign parts the chief are Mentone, San Remo, Pisa, Rome, Cannes, Algiers, and Corfu. Some go to Harrogate and other places, on account of the mineral waters, which are useful in certain cases.

Sufficient warm clothing should always be worn, with flannel next the skin. The functions of the skin must be maintained in an active state, and a warm or hot-air bath, or even a Turkish bath, may be employed from time to time. When the weather permits, moderate exercise is advisable. The diet should be at all times nutritious, and especially if there is much emaciation. As to stimulants, no definite statement can be made; but in most cases a moderate amount of some alcoholic stimulant will be of service. The digestive organs must be attended to, and aperients administered if required.

When a severe attack of bronchorrhœa comes on, stimulants and sedatives are called for, with a hot-air or vapour bath, and sinapisms over the chest and to the extremities, or free dry-cupping. Emetics may be also employed if the fluids appear to accumulate, and cannot be expelled.

PLASTIC OR CROUPOUS BRONCHITIS.

This is a very rare form of disease, and will only require a brief notice. Pathologically it differs from ordinary Bronchitis, in that a plastic material is thrown out into the tubes, of which it forms casts. These are either solid or hollow, this depending much upon the size of the tubes in which they are formed, and they usually present a series of concentric layers; but this appearance is sometimes wanting. Their size necessarily varies according to the size of the containing tube. Usually they are confined to a limited number of the bronchial divisions, but may extend from the smallest even to the largest, though they never pass into the trachea; whereas the exudation of croup or diphtheria may even reach to the most minute bronchia. Their colour is whitish, like decolorised fibrine, but spots of blood may be attached to them. Some have regarded them as the remnants of blood poured out into the tubes, which has coagulated and lost its colour. Possibly such casts may be met with occasionally, but those now under consideration are usually, and with greater reason, regarded as the result of a true exudation on the surface of the membrane. Microscopically they consist of an amorphous or fibrillar material, in which there are exudation-corpuscles and fusiform or ovoid cells, most of which are non-nucleated, but some contain nuclei, abundant granular matter, and some oil-globules are also present.

The causes of this affection are very obscure. It is supposed to be due to some diathetic state, and is said to be sometimes associated with tuberculosis. It is most frequent in young adults, but may be met with at any age. Females suffer rather more frequently than males, and those of feeble and delicate constitution are more subject to this form of Bronchitis than those who are strong and healthy.

SYMPTOMS.—In the great majority of instances Plastic Bronchitis is a markedly chronic affection, but it has been known to occur in an acute form, particularly in infants. Though chronic in its general course, there are, however, acute exacerbations on the occasion of the discharge of the casts. The severity of the symptoms will depend upon the size of these and the degree of facility with which they are expectorated. In most cases an irrepressible hacking cough sets in, painful and spasmodic, either dry or attended with slight expectoration of ordinary characters. This is followed by dyspnœa, which may gradually increase, or come on rather suddenly. It often becomes very intense, with a sense of great tightness and oppression across the chest; and there may be an appearance of threatened asphyxia if some of the larger tubes are obstructed. Walshe has found the pulse-respiration ratio to vary from 2·2 : 1 to 3·5 : 1. The cough becomes more and more severe, and the distress greater, until particles of fibrinous material are expectorated, mixed more or less with ordinary mucus; and finally one or more masses varying in size will be expelled, which,

on being disentangled under water, will be found to present complete casts of the tubes, in the form of tree-like branchings, and having the characters already described. The cough and dyspnœa are then either entirely or partially relieved. Streaks or spots of blood are frequently seen on the outside of the casts, and occasionally on their inner surface; and there may be streaks or drops of bright blood in the mucus which is expectorated for a short time after the casts have been discharged. Copious hæmoptysis may occur before the attack comes on, but Walshe believes that the concretions are then merely altered coagula. The length of a paroxysm varies within wide limits, and it may be followed by complete or temporary recovery, or the attacks may recur at longer or shorter intervals for weeks or months. There may be an entire absence of fever, but in many cases febrile reaction sets in, preceded or not by a rigor, and it may be considerable in degree. Frequently abundant muco-purulent expectoration takes place, and extensive Acute Bronchitis or pneumonia is sometimes set up, giving rise to the usual symptoms of each affection. The general health does not suffer much as a rule between the acute paroxysms, and there may be no chest symptoms. Often, however, there is a certain amount of habitual dyspnœa, and signs of imperfect respiration.

Physical Signs.—Sometimes pulmonary resonance is in excess over a part of the chest, owing to partial closure of a tube, and the portion of lung to which it leads being over-distended with air. More commonly localized dulness is met with, owing to complete obstruction and consequent collapse. The respiratory sounds are either weak or totally absent, according to the amount of obstruction. When this is removed, the above signs disappear. Dry rhonchi, especially sibilant and a few of the moist kind, are heard in different parts. Should pneumonia or Acute Bronchitis be produced, the physical signs characteristic of either complication will be present.

DIAGNOSIS.—This form of disease may be mistaken for ordinary Acute Bronchitis, pneumonia, or pleurisy. The history of the case, the characters of the paroxysms, expectoration of membranous fragments or casts, and physical signs, serve to distinguish them. The degree of fever will also be important, and the absence of the symptoms usually met with in the above diseases.

PROGNOSIS.—It is not attended with much danger in itself as regards life, but it may lead to pneumonia, phthisis, &c., and thus cause death. Complete recovery sometimes occurs, but usually this is only temporary, the disease being one which has a great tendency to recur.

TREATMENT.—Various remedies have been recommended, but apparently their use has not been attended with much success. During the paroxysms venesection has been practised, sinapisms and blisters

applied to the chest, and various drugs administered, viz. th
ferent sedatives, tartar emetic, ipecacuanha, calomel and c
alkalies, and salines. Inhalations might be of use, and the p
should be kept in a warm room, having the air well saturate
moisture. In the intervals, Fuller has occasionally seen benefit
from the use of tartar emetic, in moderate doses, for several
Iodide of potassium and inhalations of iodine have been em
with success. The alkalies and their carbonates have also
recommended. The health must be maintained, and tonics
if necessary, more especially if there is any sign of tuberc
Quinine, iron, and cod-liver oil are often called for. A cha
a warm climate, or a long sea-voyage, might be tried; while
precaution should be taken against cold and wet. If an
threatens, inhalation of steam should at once be had recou
and persevered in.

PLEURODYNIA.

By Francis E. Anstie, M.D., F.R.C.P.

DEFINITION.—Sharp unilateral pain, greatly aggravated by respiration and other movements, in the extra-thoracic or in the intercostal muscles; unattended, except accidentally, with fever.

HISTORY.—The attack is sudden, and is usually brought on by either exposure to wet and cold, or by some rather energetic movement of the trunk or of the arm; often it is the sequel of a prolonged effort involving continuous contraction of the muscles of one side of the chest. Very commonly the patient will remember that for some days past movement of the affected part has always been irksome, and followed by aching pain. The affection tends to subside spontaneously under the influence of rest, in a few days. Often the patient has been previously subject to muscular pains in other parts.

SYMPTOMS.—The patient, after experiencing more or less preliminary soreness or aching of the part, is suddenly attacked with stitch-like pains, most commonly in the infra-axillary or infra-mammary region, and more usually in the left side than in the right. The natural play of respiration is interfered with by the severe pain which the movement causes, the expansion is therefore jerking and irregular, and the respiratory sound corresponds with this in character. No percussion-dulness, friction-sound, or other of the physical signs of pleurisy, can be detected; there is no fever, unless by accident the patient is suffering from some coincident febrile affection. *Superficial* tenderness is *not* a characteristic of Pleurodynia, but there may be dysphagia, and pain on movement of the arms.

ETIOLOGY AND PATHOLOGY.—It has been customary to class Pleurodynia as a variety of rheumatism, affecting the thoracic muscles and *their* tendinous insertions; but I can discover no satisfactory grounds for this proceeding. It appears to me that Pleurodynia is merely an intense variety of the myalgia which, in less striking forms, is very much too common, and besets far too large and miscellaneous a class of patients, to allow us for a moment to assume that the rheumatic taint is a necessary factor, or indeed a factor at all. I have

several times seen very severe Pleurodynia in patients whose history showed no trace of rheumatic tendencies; and on the whole there seems to be far better evidence for the connexion of this malady with the neurotic than with the rheumatic constitution. In the absence of any sufficiently accurate and extensive statistics, I must provisionally believe that the exciting cause of Pleurodynia, like that of myalgia generally, is over-long or over-severe exertion of a muscle in proportion to the state of its nutrition, and that the predisposing cause is the neurotic constitution.

As regards the intimate pathology of Pleurodynia we know little. There is nothing to point out any special anatomical condition of the affected muscles as a constant attendant of the malady except this, that Pleurodynia occurs, for the most part if not always, in persons with slight and thin muscles, suggesting under-nutrition of these structures. I can see no shadow of reason to suppose that a local *inflammation* has anything to do with Pleurodynia; and the results of treatment are directly opposed to such an idea.

DIAGNOSIS.—This is the really important aspect of Pleurodynia. It is extremely likely to be mistaken for pleurisy, thus causing alarm, and, in the hands of some practitioners, a disastrously heroic treatment. The total absence of alteration in the pulse-respiration ratio, and of the physical signs of pleurisy, must soon undeceive any one who is even moderately careful; but during the first few hours the ablest practitioner may be at fault. This is especially the case in two situations: first, when the malady accidentally coincides with a catarrh, or some other affection causing feverishness; secondly, where the patient is a highly nervous person, whose circulation is habitually much accelerated by pain or any other cause of distress. Such being the fact, it is the more fortunate that the modern treatment of pleurisy no longer includes those heroic measures by which it was once the fashion to attempt to cut short the disease at the outset.

PROGNOSIS is scarcely worth mentioning. The affection is trivial, and certain to yield in a few days at most.

TREATMENT.—Two remedies only are necessary. The side should be covered with a sheet of spongio-piline or with flannel and oil-skin; or a simpler and readier method is to surround the side with a piece of thin macintosh, which may be put on over the flannel shirt, jersey, or spun-silk vest. One quarter of a grain of morphia should be subcutaneously injected, and repeated if necessary, in two hours' time. This plan never fails to give complete relief, but the patient should be sedulously warned against all movements not absolutely necessary, for a few days after the pain has ceased.

PLEURISY.

BY FRANCIS E. ANSTIE, M.D., F.R.C.P.

DEFINITION.—Inflammation, partial or general, of one or both pleuræ, attended with the effusion of lymph, lymph and serum, or pus.

HISTORY.—The circumstances under which Pleurisy may arise are very various ; but a practical line of distinction separates two main varieties of the disease. Pleurisies may be divided into *Primary* and *Secondary*.

By Primary Pleurisies we mean those in which the cause of the affection operates directly or mainly upon the pleura itself ; and the inflammatory affection of that serous membrane arises, so to speak, *in* a time of health, and only secondarily implicates the rest of the body, by means of the constitutional fever which it excites, or by some other results, mechanical or physiological, of the local disease.

By Secondary Pleurisies we mean those cases in which the pleural inflammation is a complication, or a secondary production, of some other visceral disease, or of some constitutional malady which has gained a hold upon the organism. Even this classification may require to be remodelled at a future day ; we may possibly find it to be too absolute : but it appears to correspond well with the facts as we know them, and it marks out in a convenient manner some broad features by which two kinds of Pleurisy are distinguished in the important matters of vital significance and appropriate treatment.

Far less practical is the attempt to divide pleurisies into *acute* and *chronic :* at least it is only in discussing the strictly clinical aspect of the disease that we can say anything useful under this heading. Two facts which are the eminently characteristic results of modern investigation have mainly tended to supersede the division into acute and chronic : first, the increasing certainty that primary acute pleurisy is but rarely fatal ; and secondly, the discovery that those chronic cases which are merely the prolongation of the acute primary variety, both may and ought to be treated with a boldness and energy which tend greatly to abridge the course they would formerly have been allowed to run. With modern means and maxims of treatment, it is not too much to say that primary chronic pleurisy has lost its most important features and its peculiar terrors : and the only reason for

regarding Pleurisy of chronic type in any special way is the fear that underneath the apparently merely local affection there may lurk the taint of, or the tendency to, a constitutional disease like tuberculosis.

Of the acute primary disease, in robust subjects, the history is essentially this. It attacks suddenly, lasts from ten days to three weeks, and then, in the majority of cases, departs, leaving behind it no other than trifling local changes, which are of no injury to the patient's subsequent health and activity. In a smaller number of cases, however, it produces an amount of effusion which is with difficulty got rid of, and unless evacuated by surgical means may remain, and protract the state of ill health, for many weeks longer. In such cases, also, the amount of permanent mechanical danger to the organs may entail disastrous after-consequences; or it may even happen that constitutional disease of a fatal kind (especially tuberculosis) may be secondarily set up.

Of Pleurisies that are "chronic" throughout—*i.e.*, that commence in an insidious manner,—by far the greater number are not primary, but secondary to some constitutional malady or some disease of another viscus. Nevertheless, it unquestionably happens, in a few cases, that apparently healthy persons are attacked with pleuritic mischief of so insidious a kind, that almost before the patient knows that he is ill (although he may have been slightly ailing for some days or weeks), it is discovered that one pleura is half or three-quarters full of fluid. Such cases are commonly very tedious in their course, and, if they do not compel the performance of paracentesis at first, take the form of an empyema or collection of pus.

When we turn to the consideration of secondary pleurisies, we find a far greater variety of type, and a much more serious prognosis, attaching to these maladies. To speak first of the Pleurisies which come in as a secondary complication of acute fevers. The whole type, and the vital significance of this class, depends on two factors—the virulence of the original disease, and the power of resistance which the organism has so far presented to it. That a patient with typhoid fever, or acute rheumatism, for instance, is suddenly attacked with Pleurisy, may be of the greater or less consequence, according to the amount of vital power of resistance which the tissues generally, and the organs of vegetative life (especially the heart and kidneys) retain. The main points, however, which the history of the pleurisies secondary to acute fever presents, are the protracted course, the tendency to purulent character of the effusion, and the frequent termination, either in death or in disastrous results in the way of constitutional disease, especially tuberculosis.

The pleurisies produced by *pyæmic infection* are mere incidents of an almost necessarily fatal blood-poisoning.

Far different is the history of the pleurisies which are secondary to the common form of *pulmonary phthisis*. The great majority of these take the shape of acute and strictly limited fibrinogenic inflammation, and, unless very injudiciously treated, tend to rapid termination,

with no worse result than a local adhesion of the pleural surfaces. More must be said on this point hereafter; at present it will be enough to state that until the later stages of pulmonary phthisis, it is decidedly uncommon, in my experience, to see pleuritic attacks causing considerable serous or sero-purulent effusions, unless they are "actively" treated, in the sense of a free use of depressing remedies. But when once a patient, with an already considerable development of chronic destructive lung disease, has acquired, in addition, a large serous or sero-purulent effusion in his pleura, the chances are heavily in favour of a disastrous termination of the disease, and it may even happen that a swift development of true tuberculosis may carry the patient off in a very few weeks, though the pleuritic effusion were, in itself, quite incompetent to endanger life.

The pleurisies which are consequent on acute or sub-acute diseases of other viscera are of very various types. Pneumonia, for instance, is in numberless, perhaps almost in all cases, attended with a certain amount of fibrinogenic pleurisy: but fortunately this is, in the majority of cases, limited to the production of a circumscribed effusion of lymph, which leads to no serious results. It is far otherwise with the more infrequent cases of pneumonia, which become complicated with effusion of a considerable quantity of pleuritic fluid: this form of secondary pleurisy usually presents acute and highly dangerous symptoms at first, and if not rapidly fatal (as it often is) is usually intractable in its after-course.

The form of Pleurisy which is secondary to Bright's disease, is always a grave and intractable affection: but its history differs greatly according to circumstances. Where it is the immediate consequence of the acute albuminuria of scarlet fever, the tendency is towards a rapid change of the effused fluid into pus; and the mildest result probable is a chronic empyema, with too often fatal secondary results. A different type of Pleurisy may be seen occurring as a complication, often a late one, of the cirrhotic or contracting form, or (much more rarely) of the amyloid form, of renal disease. In neither of the two latter forms is there the same tendency towards the rapid production of pus, but rather a tendency towards the effusion of a large quantity of fibro-serous (chiefly serous) effusion.

As for the pleurisies said to be secondary to acute cardiac disorders, it may well be doubted whether these are not always to be considered as results of some blood-poisoning, or constitutional vice, to which the heart affection is also due. Their course depends upon the degree of vital power which the organism has retained in its struggle with the constitutional malady. They can only be considered as incidents in a more formidable disease. It is, however, an open question whether pericarditis may not excite Pleurisy by direct extension of the inflammatory process.

ETIOLOGY.—Upon the etiology of Primary Pleurisy we possess no sure information at all. There is, indeed, a limited class of cases in

which the inflammation is the direct result of a blow or some other injury; but we know of no other causes, properly so called. Among exciting causes, *cold* has often been confidently stated to be a frequent one; but some of the best authorities of late years entirely deny this; and Ziemssen, out of 54 cases of Pleurisy of which he minutely examined the history, could not trace the disease to exposure to cold even in a single instance. I have myself had some reason to think that extreme muscular over-exertion and exertion in continuous public speaking produces Pleurisy, sometimes, in otherwise healthy persons.

Of predisposing causes, age has been reckoned an important one. It was supposed by some that it never occurred in very young children; *e.g.* Barrier formally denied its occurrence at all in children under six years of age. It is difficult to ascertain the exact degree of frequency of Pleurisy in young children, because in them the disease is particularly likely to occur without being detected. But all the best authorities now agree that Pleurisy is quite common among children —at any rate, after the first year of life; and Guinier, of Montpellier, actually tapped an empyema in a child twelve months old. Steiner and Neuretuer,[1] in a noteworthy series of papers, express the opinion that in young children Pleurisy with liquid effusion is the rarer; Pleurisy with proliferation of connective tissue the more common. Ziemssen[2] tabulates the ages of 54 children whom he treated for Primary Pleurisy :—First year of life, 3 ; second, 1 ; third, 7 ; fourth, 4 ; fifth, 1 ; sixth, 4 ; seventh, 4 ; eighth, 5 ; ninth, 9 ; tenth, 7 ; eleventh, 2 ; twelfth, 1 ; thirteenth, 1 ; fourteenth, 2 ; fifteenth, 1 ; sixteenth, 1. This very interesting record sufficiently disposes of the idea that there is any immunity in infancy.

A similar investigation of Ziemssen seems to show that there is no well-marked influence of *seasons of the year* as a predisposing cause.

Of Secondary Pleurisies, the exciting causes are numerous. Among the fevers, scarlatina and typhoid are especially notable in respect of frequency of occurrence, variola in regard of danger ; acute rheumatism is a frequent cause; alcoholism and pyæmic poisoning often produce Pleurisy, *inter alia.* Of tuberculosis proper and catarrhal pneumonic phthisis, it may be said that they frequently act as predisposing, and frequently as exciting, causes of Pleurisy. Of diseases of other viscera pneumonia is the most common cause of Pleurisy; after this comes kidney disease, which is, at any rate, a very powerfully predisposing cause; finally, any organic disease which necessitates mechanical pressure on, or irritation of the pleura : and it is possible that inflammation now and then passes over to the pleura, by mere contiguity, from neighbouring parts, *e.g.* the pericardium.

CLINICAL HISTORY.—The symptoms of Primary Pleurisy of acute type are as follows :—The patient, after suffering for a variable number of hours, or hardly suffering at all, from general malaise and loss of

[1] Prag. Vierteljahresch. 1864-65 ; Schmidt's Jahrbuch. 129, p. 189. The papers are part of a series of "Clinical Records of Children's Diseases."
[2] Ziemssen, Pneumonie und Pleuritis im Kindesalter.

appetite, is attacked almost simultaneously with sharp stitch-like pain in some portion of the thoracic wall (by far the most frequently in the anterior or the lateral portion, a little below the level of the nipple), and with more or less shivering. The face is generally pale and contracted with the lines of pain; the patient bends over towards the affected side, and draws his breath with visible difficulty, in a hurried, uneven, and shallow manner (*respiration entre-coupée*). After this has lasted a short time flushing of the face appears,[1] the pulse rises in frequency, and the general phenomena of pyrexia are evident; in some cases the pain now greatly diminishes, in others it maintains its intensity.

The frequency of the pulse in the early stages of Pleurisy varies considerably. There are plenty of slight cases of localized fibrinous inflammation in which hardly anything like pyrexial rapidity of pulse is present; the frequency may not be more than 86 or 90, and I have even seen a case in which it never rose above 80. In cases of primary fibrino-serous Pleurisy the pulse-frequency may be said to vary between 90 and 120 in the stage of febrile reaction after the initial rigor; on examining the notes of twelve such cases, I find the average rate at this period was 99. It must be said that all these patients were adults, and that a considerably higher pulse-rate may be found in young children, though this is by no means always the case.

The *quality* of the pulse is a point which I have particularly investigated in a considerable number of cases, and it seems to me quite certain that this follows a uniform course on the whole, regard being had to the general vital status of the patient. In the first stage of acute pain, with more or less tendency to shivering, the pulse, as tested with the sphygmograph, presents the " algide " form, *i.e.* the pulse-waves are very small and nearly devoid of secondary markings. As soon, however, as flushing of the face occurs, and a general sense of burning heat of the skin, the pulse passes to the true pyrexial type; the waves become large and dicrotic. One reads constantly, in standard works, of pleuritic patients with (sensibly) hot skin, flushed face, and a *hard* bounding pulse: but the sphygmograph, in my belief, destroys this clinical picture, for it uniformly shows that the large and somewhat bounding pulse is always decidedly *less resistent* than that of health.

The *temperature* follows no regular course in Pleurisy; in the primary disease we rarely derive any useful indications from it. On this point I agree, in the main, with the conclusions of Wunderlich,[2] and I shall say more about it when treating of Prognosis. It is enough here to say that temperature-changes keep no sort of parallel with the pulse or the respirations.

The respirations in acute Pleurisy are both absolutely rapid, and

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[1] The flushing is never so fixed and deep a colour, and especially never so markedly one-sided, in pure Pleurisy as in pneumonia.

[2] Das Verhalten der Eigenwärme in Krankheiten. 2te Aufläge. Leipzig, 1870, pp. 374-6.

especially so in comparison with the pulse ; the rapidity being mainly due to the impossibility (from pain and soreness) of taking deep breath.

Cough is a very usual, though not universal, accompaniment of the acute stages of Pleurisy. It is short and hacking ; and is either perfectly dry or attended with only a moderate amount of thin mucous expectoration ; except, indeed, when the Pleurisy is complicated with pneumonia.

The *decubitus* has been made a strong clinical feature by some writers on Pleurisy ; but there are contradictory statements, and from my own observation I should say that there is no attitude characteristic of the disease, except that which very generally prevails in the first acute agony, viz. half lying, half crouching, *on the affected side.* The decubitus is frequently changed two or three times in the course of the illness, and, except as attracting our attention to the physical examination of the chest, is seldom of any moment.

Along with these phenomena there is a variable amount of nausea, white-coating of the tongue, thirst, and anorexia ; the last usually complete.

It must be observed, however, that the above is only the picture of the early stages of a *typical* acute case in an adult. Even in adults there may be, in cases that run a pretty severe course, scarcely one noticeable symptom to arrest attention in the early days of the malady.[1] And in children the febrile symptoms, particularly the initial rigor, are often inconsiderable, and the cough scarcely attracts notice, especially in slight cases.

Physical Signs.—It is to these that we specially direct attention when suspicion of Pleurisy exists, and the information they afford is more valuable than any other.

Inspection.—When the chest is laid bare, it will be seen, in the very early stages, that the pleuritic side of the chest is somewhat retracted, and its intercostal muscles nearly or quite motionless, while increased play and movement of the sound side is observed. At a later period of effusion a positive dilatation of the pleuritic side and a bulging of the intercostal spaces may often be noted, but it is possible, even in cases of very extensive effusion, for the chest wall to remain apparently unaffected, the force of displacement being spent mainly on the neighbouring organs. But in all cases where there is the least suspicion of Pleurisy, accurate *repeated* measurements of both sides must be adopted :[3] it will not do to trust the eye, for enlargement of the side may be obscured by the general configuration of the thorax, or it may happen that the expansion of the *sound* side (in its compensatory efforts of breathing) may assist in concealing the fact. One of the most striking pieces of evidence offered to the

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[1] See, among other authors, Trousseau (Clin. Médicale, tome i.) for some striking examples.

[2] Ziemssen, Pleuritis u. Pneumonie im Kindesalter.

[3] Cf. Verliac (Épanchements pleuritiques, Paris, 1865), particularly case at pp. 19, 20.

eye is the visible displacement of the heart which usually presents itself when the effusion is large : in the case of left pleurisy, the apex will be seen beating under or to the right of the sternum, or in the epigastrium ; in right pleurisy it may be found beating to the left of the left mammary line, and, in extreme instances, even in the left axilla.

Mensuration.—In the early stages there is commonly no enlargement of the affected side ; the sound side, indeed, appears, and is, the most expanded. But as effusion comes on the balance is restored, and when the fluid becomes copious the intercostal spaces yield, the ribs become more separated, and in proportion to the yieldingness of the thoracic wall a real increase in the size of the affected side is observed. It is only in the slight-made chests of young children that this is early perceptible ; in adults, the displacement is rather on the side of the viscera until the effusion becomes very large. Strict daily comparative mensuration of the two sides ought nevertheless to be practised from an early stage.

Percussion yields no information in the first stage, nor can it be well tolerated. Supposing the case to be one of merely fibrinous exudation, we may get, from first to last, scarcely any abnormality in the sound elicited ; but the chest wall gives a strange sense of deadness and inelasticity to the percussing finger. Very solid and thick fibrinous deposits may cause a really dull percussion sound. When serum is poured out in any quantity, however, the evidence from percussion becomes striking : over the whole space occupied by the fluid there is found a dulness, more pronounced in some cases than in others, but always with a character of its own, which must be heard to be recognised, but is much more marked than that produced by lung-consolidation. In ordinary cases, where the fluid is not bound and localized by adhesion, the dulness reaches upwards from the base of the chest to a variable height, according to the amount of the effusion : its character is very perceptible in comparison with the sound side, but the line of its termination above is by no means always an evenly horizontal one. As the case proceeds, and an increasing quantity of serum is effused, the dulness may extend quite up to the clavicle in front and to the supra-spinal fossa behind ; after this, any further extension of the effusion necessitates expansion of the pleural cavity in some fresh direction. So far, space for the fluid has been obtained chiefly by the compression of the lung into the spinal fossa, but already, in most cases, there has been also displacement of the surrounding organs. This displacement may affect chiefly the ribs. But the diaphragm even more certainly yields, and its displacement downwards pushes the liver down (in right pleurisy), and percussion can recognise the depression of this organ ; similarly, the stomach and colon may be recognised, by their tympanitic percussion-sound, at an unusually low level, and the spleen-dulness may sometimes be traced at a point below the margin of the right false ribs (in left pleurisy). The most striking phenomena of displacement

are, however, connected with the heart. In left pleurisy a large effusion often pushes the heart so much to the right that the cardiac percussion-dulness is found to occupy a space beneath the lower end of the sternum, and even extending considerably beyond its right border. The displacing effect of right pleuritic effusion is less immediately obvious to percussion; though readily identified by inspection and palpation, or at any rate by the stethoscope.

Palpation often gives us very important information. In early stages, and in those cases where the effusion remains merely fibrinous, the grating of the lymph-covered pleural surfaces may communicate a thrill to the chest-wall which is appreciable by the hand; these phenomena, however, are not very frequently observable. More constantly useful is the absence of vocal fremitus when liquid has been poured out in any quantity; this is usually striking when we compare the pleuritic with the sound side. The fremitus of lymph-covered pleural surfaces may sometimes be felt in the later stages after the fluid effusion has become absorbed.

Auscultation rarely reveals much in our earlier examinations. Fluid has not yet been poured out. We may happen to catch the moment when the pleura is rubbing its two fibrine-covered surfaces together at some point or points, in which case the " friction-sound" is heard accompanying inspiration and expiration. There is no use in attempting to describe this sound minutely; it does, in fact, considerably vary in pitch and in character; the student must himself repeatedly hear it, and compare it with other sounds (especially various *clicking* bronchial sounds) before he can identify it with confidence. The rarity with which, as I have said, it is heard in the early stages, holds good in the ordinary type of Pleurisy that goes on to liquid effusion; and in the wards of hospitals this is the prevailing type of the disease that is seen; but I have been for some years past surprised at the frequency with which I have detected slight and limited Pleurisy, by means of the friction-sound, in the out-patient room. Most of these cases were tuberculous; but a considerable number afforded no room for any suspicion of the kind. The friction-sound is far more commonly heard in the stage of resolution, where liquid is getting absorbed, and the roughened pleural surfaces come together again.

In the stage previous to fluid effusion, the ear detects only the fact that the lung of the healthy side is expanding more vigorously and noisily than the other. When fluid becomes effused, however, the tendency is at first often towards a bronchial character of the breath-sound, accompanied by bronchophony on the affected side, while the effusion is small. In adults, however, the progress of the effusion rapidly replaces this by weakening, and finally absence, both of breath and voice sounds; meantime the breathing on the healthy side is more and more noisy and puerile. On the pleuritic side, the lung getting pushed back into the spinal fossa, there are bronchial breathing and bronchophony to be heard in the upper and inner scapular region

between the scapula and spine, and comparative or complete
e elsewhere.

ægophony, the curious bleating sound of the voice which is
imes heard at the upper level of the fluid, I feel inclined to
ery little. It is in truth one of the *fancy* signs of Pleurisy—
sting rather than useful; it is so inconstant, and there are so
fallacies attending its recognition, that I believe it to be, for
ary auscultators, rather a snare than a help. In a similar way
ust speak of the *succussion sound*, the splashing noise supposed
heard on shaking the patient; this also is very inconstant.
we may here notice that the changes both of voice and breath-
s, and also of percussion sounds, which are commonly supposed
induced by *changing the patient's posture*, are very uncertain
unreliable in true Pleurisy.

scultation is of great value in indicating the altered position of
eart, which occurs in cases of large effusion.

ysical *Signs in Pleurisy of Children.*—There are several most
tant variations from the above general picture of the physical
of Pleurisy, to be observed in the pleurisies of infancy and
ood. These variations are due to two circumstances: the small
f the chest, and the greater yieldingness of the chest-walls. As
ls the auscultation, it is all-important to note that bronchial
ing and voice persist, in nearly every case, even when the
on occupies the whole chest, and when vocal fremitus is entirely
t. Rilliet and Barthez were the first to notice the remarkable
hat even a pneumonic bronchophony and bronchial breathing, so
om being diminished, are usually much intensified by a superven-
leuritic effusion. Yet there are many text-books that take no
of this peculiarity of children, and ignorance of it has certainly
he cause of many disastrous mistakes in practice, the practitioner
ing firmly that he had merely to do with a consolidated lung,
urprised by the appearance of fluctuation and evident signs of
ing in one or more of the intercostal spaces. Another very im-
t distinction of Pleurisy in young life is the comparative *absence
ns of displacement of viscera*. The fact is that the chest-wall
more easily, and the force of pressure is not expended, to any-
like the same extent as in adults, in dislocating the heart and
iving downwards the diaphragm and the abdominal viscera.
also, is a peculiarity too little noted in text-books written by
whose experience of Pleurisy in children is not large; and,
l with the persistence of bronchial breathing and voice, has
less caused numbers of mistakes. Such errors probably cost
ves of many patients who might have been saved by prompt
g. It is, however, a mistake to say, as some have done, that
cement of viscera never takes place in children. Ziemssen [1]
s a conclusive series of cases, observed by himself and others,
contrary effect.

[1] Op. cit. pp. 67, 68.

III. 3 o

We must now complete the clinical history of Pleurisy b scribing what may be called its critical symptoms. If the case the turn towards recovery by simple absorption, which is the n destiny of primary Pleurisy, then, after the subsidence of p there occurs, usually, a pause of a day or two, after which th of absorption begins to show itself by physical signs, and by a but increasing degree of relief to respiration. Among the sign carefully to be looked for as indicating the commencement process is the return of the percussion dulness of the liver, tympanic sound of the stomach, to a higher level. The return heart to its proper position, even when absorption has made co able progress, is not always rapid or at first very evident. accompanying sketch is represented, with rough but su accuracy, the state of things in the chest of a young but intem
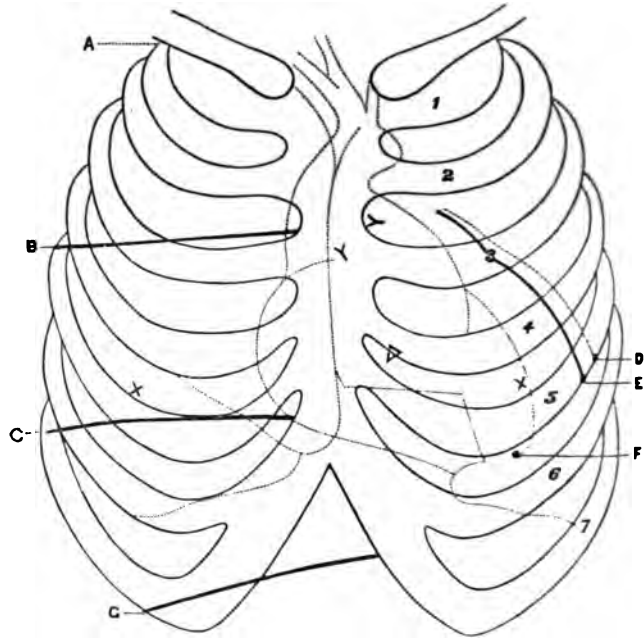
A. Line of original upper level of fluid ; B. Line of fluid on twentieth day ; c. tural upper level of liver; G. Termination of dulness below (tympanitis D. Maximum heart-impulse at time of greatest effusion ; E. Ditto on t day ; F. Ditto in natural state.

man, C. J., who, between the sixteenth and twentieth days o Pleurisy, experienced an amount of absorption of the effuse indicated by the distance between the lines A and B. The liv believed to be somewhat enlarged and fatty in this man.

Instead of a speedy commencement of absorption, the flui

remain in a passive condition, and the patient may continue in a state marked by no discomfort except some mechanical embarrassment of respiration and heart movements, impeding him in any but the gentlest movements. It is fortunate, but comparatively rare, when a protracted period of this kind is terminated by the occurrence of absorption. More commonly a slight but steady increase of ill-health is experienced, till at last there arises decided febrile disturbance, settling more and more into a hectic type, with copious sweat, morning remissions and evening exacerbations, and, in short, a more or less complete series of indications of extensive suppuration. It is here, at last, that the thermometer, so little to be depended on in other stages of Pleurisy, often gives us precious information of the changed aspect of affairs.

There is no need to carry the clinical description any further, since under the heads of Prognosis and of Treatment sufficient information will be found concerning the favourable and unfavourable termination of suppurative Pleurisy.

Complications and Sequelæ.—Of primary Pleurisy the most frequent complication is pneumonia, and this may either exist from the first or supervene at any tolerably early stage. It does not appear to occur with any frequency after the lung has been compressed into a small space by fluid exudation. Laennec believed that the compression by the fluid always tended to prevent the occurrence of *severe* pneumonia in connexion with Pleurisy; but it will be seen, under "Prognosis," that he was at least wrong in this, so far as relates to *children*. But when the lung is compressed to carnification, it is doubtless very incapable of inflammation. The most formidable way in which pneumonia may complicate Pleurisy is where, a considerable effusion existing in one pleura, inflammation attacks the *opposite lung*. It may be doubted, however, whether this ever occurs in truly primary Pleurisy: personally I have never seen a case where inquiry did not show the existence of kidney disease, fever, pyæmia, or some of the many causes of secondary Pleurisy.

This is the place to speak of double Pleurisy, which may fairly be looked on as a complication; and in regard to it I can only repeat the same observation. Primary Pleurisy, as we call it, does seem, at any rate, peculiar in this—that it is an essentially unilateral disease: and I have never been able to see a double case in which there were not ample reasons for thinking the Pleurisy a secondary affection to some condition of general blood-poisoning. It is in the same point of view that we must regard the supervention of other serous inflammations, *e. g.* peritonitis; but there is a possibility, perhaps, that *pericarditis* may sometimes arise by simple extension of the inflammatory process from the contiguous pleura.

In cases of empyema of some standing a not very uncommon complication is discharge of the pus through a pulmonary fistula into the bronchi; this is associated with phthisical lung disease in a large majority of cases, but a considerable number are recorded in which

the accident has occurred in primary Pleurisy without tubercular disease.[1] The cases are rare in which the channel of evacuation has proved sufficient : usually the bronchial discharge is only a preliminary to subsequent perforation outwards, and as regards treatment this is the view that should be taken. The accident of pulmonary perforation must be looked on as the probable commencement of a period of *fetid suppuration* and pyo-pneumo-thorax.

Of the sequelæ of Pleurisy one outweighs all others in interest, viz., *tuberculosis.* It is now well established, not merely that Pleurisy often occurs in phthisical lung-disease, but that Pleurisy itself is capable of setting up true tubercle, even in previously healthy persons. This is specially apt to occur where a purulent effusion has been allowed to remain too long in the pleura, or where paracentesis has been performed repeatedly for empyema, the wound being closed in the interval. But the latter practice is one which, it may be hoped, will no longer be followed.

The other sequelæ of Pleurisy, though they may be very troublesome, are less important. Retraction of the chest-wall and consequent deformity of the spine and shoulder is the ordinary result of the absorption of a large effusion, where the lung has been too much bound by adhesions to re-expand at once, or perhaps at all. The same thing occurs where a collection of pus has been allowed to burst externally ; here the lung is firmly bound down, and the orifice of the rupture being valvular no air enters the chest, and so the ribs sink in under atmospheric pressure. It may be at once said, however, that these deformities are merely temporary, and that with proper attention they will always be found to right themselves, in the course of a year or two, almost entirely ; this is especially the case in children. As regards the fistulous opening left after the natural bursting of an empyema, the course of events depends on the amount of local mischief which was done during the passage of the pus to the surface ; when this has been very tedious, more or less extensive destruction of periosteum and necrosis of ribs may occur, and may give much trouble. A single fistulous opening is merely to be looked at as an unpleasant fact which will disappear in a certain number of months or years.

PATHOLOGICAL ANATOMY.—The first stage of change in every case of Pleurisy appears to consist of ordinary injection of the vessels beneath the pleural membrane ; in primary cases by far the most frequently this change begins in the costal pleura. Slight ecchymoses are more or less plentifully scattered over the hyper-vascular and bright-red part. The clear serous membrane also begins to be clouded and swollen, and if the inflammatory process goes on for a very few hours, there occurs a visible deposit of fibrinous lymph, of a reddish-yellowish tinge, and at first very tender and soft, and small in

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[1] For a good discussion of the subject of bronchial fistula see Aristide Attimont, "Résultats de la Paracentèse dans la Pleurisie purulente," Paris, 1869.

quantity. If the inflammation goes on to be an affair of more than a day or two, not only does the amount of fibrinous deposit increase by successive layers, but a variable proportion of serosity is mixed with the lymph; and often serum is poured out in large quantity from an early period, so as to fill a large portion of the pleural cavity within a few days, more rarely within a few hours. There is great variability in the relative amount of the serous and fibrinous elements of effusion, but in general the contents of the inflamed pleura may be described as consisting of yellowish serum in which float a quantity of concrete masses of the same fibrinous matter which lines the inflamed portion of the membrane; and, as the case advances, bands of fibrinous matter, at first tender and yielding, afterwards firm and tough, form adhesions between the costal and the opposite portions of the pulmonary pleura. In some instances it happens that the fibrinous adhesions are so many and so dense over a limited area, that they enclose and limit the serous exudation, confining it to a comparatively small portion of the pleural cavity. In ordinary cases the pleural cavity becomes progressively and more or less evenly filled to a higher and higher level, the lung receding before the fluid, and being pushed upwards, backwards, and inwards, till it is compressed into the mediastinal or spinal fossa. On the other hand, it may happen that a comparatively small amount of fluid spreads itself rapidly over a large portion of the lung, and, though reaching a high level in the chest, does not greatly compress or alter the position of the lung, at any rate at first.

In those pleurisies where the inflammation is limited to a small area, the effusion often consists almost entirely of plastic fibrinous matter, and then the regular course of the affection is short, ending in an adhesion of a limited portion of the opposed pleural surfaces. Undoubtedly the most frequent examples of this kind are found to occur in the course of chronic pneumonia and of phthisis; but it is certain that they also occur, sufficiently often, in individuals who are otherwise apparently healthy.

Another outcome of inflammation is the effusion of pus, which may either exist from an early period or may slowly develop in the course of an ordinary fibro-serous pleuritic effusion, the pus-cells more and more invading the serosity, until at last the whole mass of fluid assumes a truly purulent character. Pus in the pleura is known under the name of empyema. *Acute* empyema is rare as a primary disease in adults (more common in children), and is usually the direct result of *injury*, but is common enough as a complication or sequela of certain acute fevers, especially scarlatina, and also pyæmic poisoning; it occurs also in a certain small number of cases of pulmonary phthisis; and one special variety—pyo-pneumo-thorax form of perforation of the pulmonary pleura—is in such a large proportion of instances due to chronic lung-phthisis, that it may for practical purposes be almost entirely left for consideration along with that malady.

The fibrinous element is, I believe, never really absent from a genuinely pleuritic effusion: many times as I have looked for such a thing in the post-mortem room, I have never seen a purely serous nor a purely purulent Pleurisy. [The cases of miscalled purely serous Pleurisy are always merely hydro-thorax, occurring either as a mechanical result of embarrassed circulation, or else as a consequence of poisoned or depraved blood.] The extent to which the fibrinous element exists varies from a slight coating of soft lymph upon limited portions of the pleura (both costal and pulmonary), with some light floating flakes in pretty clear serous fluid, to a dense cortex enveloping the whole of the lung and coating the whole of the pleura, and from one to several lines in thickness; the latter condition is only produced in old-standing cases, and the lymph is deposited in concentric layers of which the external are yellow and rather soft, the deeper ones dense and tough, reddish in colour, and exhibiting traces of vessels. The longer the pleural cavity remains distended with fluid, the more firmly the lung is bound down to the neighbourhood of the vertebral column; and if the conditions of mechanical pressure last long enough, the fibrous adhesions grow too dense ever to be removed so as to allow the lung to expand again. The final result, in cases of recovery, is the conversion of the layer of fibrinous lymph into a rudely organized cellular tissue, bands of which also stretch between the lung and the chest-wall, and either bind them firmly together, or (according to their length) allow more or less free play. In exceptional cases the whole surface of the lung is left firmly adherent to the chest-wall by a layer of fibrinous matter, which may vary in texture from that of loose cellular tissue to a tough semi-cartilaginous material. The latter condition has been occasionally seen in cases where, apparently, there has been little or no serous effusion, and where the lung, though thus universally coated, has been found (after death from some other disease) fairly expanded and permeable to air.

If we now inquire into the minute anatomy of these changes, we find that the earliest stage, beyond that of mere congestion of the sub-pleural vessels—that, namely, of cloudy swelling of the membrane—is microscopically distinguished by the appearances of proliferation of the epithelial cells, which tend more and more to multiply and also to be shed from the surface.[1] Very soon there appear, also, masses of fibrinous materials which have nothing to do with epithelial changes, but apparently exude directly from the blood-vessels, and belong to the same process by which the serous effusion is poured out. The proliferated epithelial cells, together with exuded blood-cells, form the cellular element of the fluid; and upon their numbers and the stamp of their vitality depends the question whether that fluid shall turn to pus or not; they are present, along with fibrinous matter, in the flocculi which float in the fluid.[2]

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[1] Rindfleisch, Handbuch der pathologischen Gewebelehre; Leipzig, 1869.
[2] See the striking engraving in Rindfleisch, op. cit. p. 211.

As to the retrograde changes which take place in the solid matters when absorption takes place, the most important matter is this. If the cellular elements are in large quantity, the retrograde process is slow, and passes through a stage of cheesy formation, which may be very lingering : and there is much probability that this state, though not so *frequently* as the long-continued persistence of a purulent effusion, may give rise to tubercle. Where a very abundant and thick fibrinous deposit, with scattered cells, is the only thing left after the liquid has disappeared, there is still some danger : part of the material must pass through the stage of caseous formation : and it will be well if the patient escapes with a thick, almost cartilaginous, coating of his lung, scattered with calcareous deposits. The most favourable result is when the only trace of the effused matters is a few adhesions, composed of cellular tissue, between the lung and the chest-wall. Such appearances are, as is well known, among the commonest things found after death in the pleura even of persons who may never have been conscious of the pleuritic attacks at the time of their occurrence.

The condition to which the lung is reduced by the pressure of the effusion and the strangulation induced by the compressing fibrous adhesions, is of the highest importance. In simple Pleurisy, without pneumonic complication, the lung, pressed back by the side of the spine, is reduced to a state of so-called *carnification;* the tissue is hard and unyielding, and does not crepitate under pressure. This is the effect of extreme compression ; when the effusion has been small, the lung-tissue may still be partially crepitant. Even the extreme degree of carnification does not seem to exclude the possibility of re-expansion, if once the pressure were fairly taken off. The danger is rather—especially when the fluid has been purulent—that cheesy masses, and even true miliary tubercle, may develop within the compressed lung. It is in these conditions that the re-expansion of the lung becomes almost beyond hope.

On the other hand, when there has been antecedent pneumonic inflammation, the lung may never become greatly compressed from first to last ; it will be found heavy and solid, much less resisting to the finger than true carnified tissue, perhaps still crepitant, but presenting the characters of hepatization.

DIAGNOSIS.—The most frequent source of fallacy is confusion between Pleurisy and pneumonia.

In both diseases there are fever, dyspnœa, and cough. But in primary Pleurisy the temperature rarely attains a high grade, especially in early stages ; while in pneumonia it is not unusual for the thermometer to reach 103° or 104° within the first twenty-four hours. The skin is much more dry and burning to the touch in pneumonia than in Pleurisy : the flush on the face more fixed, and often remarkably unilateral. The feeling of dyspnœa is often much more remarkable in Pleurisy than in pneumonia, but the relative frequency of respiration and pulse is more altered in the latter. The cough in Pleurisy is short

and hacking, but attended with no expectoration or with only the discharge of a little thin mucus; whereas in pneumonia expectoration is present in an immense majority of cases, and soon becomes "rusty" in colour, and very tenacious. Sharp stitch-like pain in the side is a very frequent characteristic of Pleurisy, whereas in pneumonia there is commonly no pain, or else a much duller and more diffused pain. As regards physical signs, the dulness on percussion is more absolute in Pleurisy than in pneumonia, and as the case proceeds the breath-sounds and voice become weakened and finally abolished in the former, while they become more and more "bronchial" in the latter. [This is true of adults, but in children bronchial breath and voice persist in Pleurisy.] The vocal fremitus becomes weakened and finally abolished in Pleurisy; it increases in pneumonia as the consolidation proceeds. Displacement of the neighbouring viscera is never seen in pneumonia; it is common in Pleurisy, especially in adults. Increase in the volume of the affected side, with widening and bulging of the intercostal spaces, and, in extreme cases fluctuation there, are characteristic of Pleurisy, but not of pneumonia.

Several of the above remarks are chiefly applicable to primary Pleurisy, which, as already stated, is nearly, if not quite, always a unilateral disease; whereas pneumonia is more frequently than not bilateral. More absolutely distinctive of Pleurisy, however, is the absence of that fine hair crepitation which in pneumonia precedes consolidation and establishment of bronchial breathing and voice. Where the chest affection is only secondary, Pleurisy is frequently double, and much of the value of comparison of the two sides is lost. Here the percussion and auscultation sounds require to be more finely appreciated; and the presence or absence of special sounds, like the fine pneumonic crepitation, is of the greatest importance. The possibility of the co-existence of pneumonia and Pleurisy must always be kept in mind; and when to fine crepitation, mixed increasingly with patches of bronchial respiration, there succeeds a weakening and then absence of breath and voice sounds, we have good *primâ facie* reason for thinking that the latter disease has supervened upon the former.

Undoubtedly the most generally serviceable physical characteristics of Pleurisy are the combination of very pronounced percussion dulness, absence of vocal fremitus, loud bronchial breathing limited to the superior internal and intra-scapular space, and (where the pleura is not yet full) tympanic percussion and almost cavernous respiration just below the clavicle, with more or less complete absence of breathing elsewhere.

The diagnosis of Pleurisy from simple *hydrothorax*—passive exudation resulting from mechanical obstruction of circulation, or in chronic blood-poisoning—rests chiefly on two facts: the absence of fresh febrile disturbance, and the more generally double effusion in the latter affection. Often there is corroboration of these indications, in hydrothorax, from the simultaneous occurrence of dropsical effusion in other parts.

Pleurisy is to be distinguished from *pulmonary phthisis* by the history of the attack, the absence of characteristic expectoration and emaciation, the physical signs of fluid effusion, the family history, &c. But as regards limited attacks of merely fibrinous pleurisy, it may be very difficult to say whether or not there is phthisis also, the form of pleuritic attack being a very common complication of phthisis at all stages.

The presence of a solid tumour, occupying a considerable portion of the pleura, or bulging into it from the mediastinum, may closely simulate most of the physical signs of Pleurisy. We must here depend mainly upon a very accurate inquiry into the history and the " rational" symptoms: the absence of all febrile disturbance at the commencement will assist our judgment.

But although I might draw out to a much greater length this catalogue of the possible snares which lie in wait for us in the diagnosis of Pleurisy from affections more or less resembling it, I think this un-necessary, because the means of discrimination are now augmented by a most potent test, the modern practice of exploratory puncture. We may fairly say that, with the assistance of the small trocar, fitted with the glass vacuum syringe, it is possible for us to make a puncture into a chest without the least apprehension of damage, whether the trocar shall enter a pleuritic effusion, a hepatized lung, a cancer, or even an aneurism; and with the great advantage of discovering whether there is fluid at all, and if so what the fluid is. It is un-necessary to say, that except under the stress of urgent symptoms, this should not be done while high febrile excitement is present, unless there is strong probability that fluid effusion is the sole cause of the maintenance of the fever.

PROGNOSIS.—The prognosis of primary Pleurisy is very favourable, though there is by no means that complete immunity from fatal consequences which was asserted by Laennec and Louis. The danger of sudden death from orthopnœa was shown by Trousseau to be a serious one in a small percentage of cases—more especially the *latent* type, with insidious commencement—when the effusion completely fills one pleural cavity; and at the present day it is generally acknowledged that this is a real peril. For prognostic purposes it is now pretty well understood that it is not the mere quantity of the effusion that should most alarm us: the rule is that, large effusion being present, the occurrence of one or more attacks of *severe dyspnœa* —*orthopnœa*—indicates a dangerous want of tolerance by the organism, and calls for direct interference.

The other danger which must be reckoned with is where a primary pleuritic effusion has remained stationary in the chest without any tendency towards absorption for a considerable period, and signs of its conversion to pus, with attendant severe hectic fever, increasing emaciation, and general prostration of vital power, show themselves. Here, the least of dangers is that involved in protracted suppuration :

far more formidable is the risk, now well established, of an infective absorption leading to tuberculosis.

It must be said, however, that both the chance of suffocation from mechanical pressure, and the risk of secondary tubercular processes, are indefinitely diminished by the modern practice of prompt paracentesis. It may be questioned whether the experience of the next twenty years will not enable us to ensure an absolute immunity from fatal results of either of these complications.

Very different is the prognosis in secondary pleurisies : though even here we may reckon on a considerable percentage of recoveries. Most fatal of all is the Pleurisy which occurs in the course of pyæmic (e.g., puerperal) infection ; here, death is the rule, recovery a rare exception. A considerably smaller mortality, but still a very high one, attends the cases which supervene on scarlatinal albuminuria ; and a somewhat similar ratio of deaths, though scarcely so high, attends all pleurisies secondary to the acute general fevers. But the fact is that every case of Pleurisy supervening on a constitutional fever has its individual prognosis, depending on the time of its occurrence, the amount of vital resistance which the original disease has spared, &c. ; and we are driven here to a minute observation of particular symptoms. It is here that thermometry plays an invaluable part. The following conclusions of Wunderlich, respecting temperature in serous inflammations generally, apply, according to my experience, with admirable correctness to pleurisies secondary to fevers : [1] " Subnormal temperatures are always highly suspicious ; death often occurs either shortly after their first appearance, or when they have persisted for some time, or have alternated with normal and excessive temperatures. Temperatures of considerable, especially of increasing height, though not necessarily in themselves of bad omen, yet add something to the dangerous momenta. If the temperature falls again, the danger is not past, yet it is less threatening than if the heat had been maintained. Besides the height of the febrile temperature, its constancy, and the absence of remissions, especially heighten the peril ; more particularly the long continuance of a high temperature, even if it alternate with considerable morning remissions. In the first case the disease is dangerous, in the latter complete recovery is at least doubtful. . . . . . Very considerable and irregular fluctuations between the highest and the lowest temperatures (resembling those of pyæmia) occur, especially in endocarditis ; occasionally also in pericarditis, pleurisy, and peritonitis ; they are always extremely dangerous, and a fatal result is very probable."

To this element of prognosis let me add examination of the pulse with the sphygmograph, of the value of which I can hardly speak too strongly. The subject would occupy too much space in discussing here ; but I would refer the reader to what I have written elsewhere [2] respecting the favourable and the unfavourable pyrexial pulse-forms,

[1] Wunderlich, op. cit. p. 375.
[2] Lectures on Acute Diseases, delivered before the Royal College of Physicians. (Lancet, 1867, vol. ii.)

and shall merely say that subsequent experience has strengthened my convictions already expressed. I believe that in the dangerous secondary pleurisies the combined use of thermometer and sphygmo-graph is more valuable for prognostic purposes than all other modes of observation put together.

As regards the prognosis of Pleurisy secondary to pneumonia, it may be said, in general terms, that the amount of danger depends entirely on the moment at which the Pleurisy supervenes. If the system has been severely tried, the chances are bad: thus, Rilliet and Barthez reckoned eight deaths out of ten such cases.

Of Pleurisy secondary to phthisical lung-disease, as already said, the prognosis is usually very favourable, for the moment at any rate: but there is always the danger that any fresh pleuritic process may be the starting-point for a true tubercular infection. And, on the other hand, the subjects of tubercle who (not a very common case) develop exten-sive Pleurisy with liquid effusion, nearly always die.

Be it said, however, that the increasing tendency to paracentesis, even in secondary pleurisies, will not improbably result in a greatly decreased mortality, even from the most formidable varieties of the disease. It is almost impossible to rate too highly the significance of such a case as that of Kussmaul, hereafter to be cited (*vide* Treatment).

There is one variety of secondary Pleurisy of which I must say a few words here, because it is scarcely discussed in the text-books, viz. Alcoholic Pleurisy. Except in the advanced stages of chronic alco-holism, supervening Pleurisy is rarely of bad prognosis: nearly always it leads only to a certain amount of fibrinous exudation and proliferation of connective tissue. It is only in the last stage of drink-degeneration that a fatal form of empyema is apt to develop itself: I have seen only one such case purely traceable to the results of drink alone, but there are a considerable number of cases in which the fatal result is, perhaps, equally due to this influence and to blood-poisoning from renal disease.

TREATMENT.—The treatment of Pleurisy is naturally divided into that of the primary and that of the secondary forms.

Primary Pleurisy, of a well-marked type, is perhaps as little the fit subject of treatment by drugs or other artificial means, in its acute stages, as any disease that could be named, or rather, the drugs needed are very few, and are all of the stimulant-narcotic class. For the vast majority of patients, indeed, the only drug which is of considerable value is opium in one or other form, until the febrile period has passed over, when preparations of iron sometimes become very useful. I do not make this statement without having carefully watched and con-sidered the effects of a number of internal remedies which are still used as a matter of course, and indeed considered essential, by various physicians of good repute.

To take, first, the case of primary simple fibrinogenic pleurisy, one may at once decide against all heroic remedies, since evidence abounds

on all sides to show that the disease is a perfectly harmless one, unless the patient has strong tendencies to constitutional disease, and that it tends always to recovery. In fact, one has no need to adopt any treatment whatever beyond keeping the patient in one room, free from draughts, and in the posture which he finds easiest to him; feeding him steadily with nutritious food of the kind best adapted to the degree of fever and digestive derangement that may happen to be present: forbidding unnecessary movements and talking: applying hot poultices to the side, and administering an occasional hypodermic injection of $\frac{1}{8}$ or $\frac{1}{4}$ grain morphia to keep the pain in check. Acetate of ammonia, in doses just short of those which produce decided sweating, will sometimes greatly relieve the pain and distress even without the aid of opium, and is at all times a harmless, even if an unnecessary medicament. Recently, the acetate of methylamine (a base which exists in roasted coffee, owing to the transformation by heat of a part of the coffeine) has been proposed, and apparently used with good effect, by Professor Béhier of Paris.[1] There is usually no necessity for alcohol, and it had better be avoided. After from six to seven days in bed, the patient will probably be well able to sit up, and the only thing necessary to forbid to him is *movement*. He should sit perfectly still. If any anæmia remains, the tincture of muriate of iron, in twenty-minim doses thrice daily, is advisable as a tonic; and, on the whole, a very few days ought to see the patient completely fit to resume his ordinary work.

In Pleurisy evidently of considerable extent, and with a notable amount of *serous effusion*, the ideal of treatment should be still, as much as may be, that given above. It is now very decidedly proved that the old heroic methods of attacking severe Pleurisy ought to be abandoned. In the first place, as to general blood-letting, I have witnessed enough of this treatment to be sure of two things: firstly, that the older physicians were perfectly right in the statement that it usually relieves *pain* with great promptitude; and secondly, that the relief thus given is not in the least degree superior to that afforded by hypodermic injection of morphia, except that it operates more quickly, perhaps by some five minutes, than the latter. As to bleeding checking the tendency to effusion, *that* is to me quite incredible. No such effect has been witnessed in either of the five cases of phlebotomy for acute pleurisy that I have watched at various times; and I observe that Dr. Aitken,[2] while still adhering to the use of this remedy, recommends us not to be discouraged by the fact that the effusion may go on increasing after the bleeding, and the patient also may feel very depressed. It is true, he says, that after a certain time absorption will set in, and that it will then go on more rapidly and well than if the patient had *not* been bled. I cannot at all imagine on what evidence this last opinion is based; certainly it utterly conflicts with the facts of my own

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[1] See a paper in the Practitioner, October 1868, "On Tonic Medication and on Acetate of Methylamine: a new Tonic Remedy." By MM. Béhier and Personne.
[2] Science and Art of Medicine, 3rd edition, vol. ii., article "Pleuritis."

experience; and though I have personally seen little of the actual treatment of Pleurisy by bleeding, I have examined a pretty large number of persons whose past history included one or more pleuritic attacks which had been so treated. The accounts given by such persons show a melancholy uniformity: long weeks and months of suffering from the presence of effusion in the chest, occasionally leading (through empyema) directly into active and rapidly fatal tuberculosis, nearly always slow and imperfect recovery, with diminished vital energy, and especial weakness of the chest, and only in the rarest cases a tolerably prompt and complete recovery. The homœopathists have made their fortunes in no small degree by their "treatment" of Pleurisy, which has had the one sole merit of being purely negative, and avoiding all destructive agencies.

A much better case, no doubt, might be made out on behalf of local blood-letting. Cupping ought never to be mentioned, being actually barbarous in the suffering it inflicts on a pleuritic patient. But leeches unquestionably do relieve pain very often in a speedy and effectual manner, and I only know of one objection to their use, viz. that morphia will relieve the pain with even greater certainty. During five years of dispensary practice I determinedly abstained from the use of leeches in Pleurisy, and found morphia, even given by the mouth, a perfectly satisfactory substitute. But since the use of the hypodermic syringe has become more common the advantages of morphia are far more manifest; and I have no doubt, personally, that leeches are now unnecessary. The first act of the physician in treating a pleuritic patient in the agony of the early acute stage, should be to inject $\frac{1}{6}$ or $\frac{1}{4}$ grain of acetate of morphia (for an adult) under the skin,[1] and to envelop the painful side in a hot poultice. For a child under 2 years, $\frac{1}{40}$ or $\frac{1}{30}$ grain is enough. Such doses as these may be repeated every four hours, if necessary; but in fact it is seldom that more than two or three doses are needed in the first twenty-four hours, and afterwards one dose in each twenty-four hours is generally enough.

I would insist strongly on the advantages, indirect as well as direct, of subcutaneous over gastric administration of opiates; in a direct way, the former is superior as acting much more rapidly; in an indirect way, because it so much less disturbs the functions of the alimentary canal.

Of the treatment by mercury I can express only the most unqualified disapproval. I have watched many cases of Pleurisy in which, according to the rule formerly acknowledged, mercury was given, either to complete or partial salivation, as soon as the signs of effusion became unequivocal, and I can truly say that these cases, even when they were not further complicated by the depressing influence of blood-letting, contrasted very unfavourably with the results of a treatment which entirely abjures mercury for any purpose except that of an occasional purgative. I am glad to cite, on this point,

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[1] I believe, with Mr. Hunter, that there is no need to inject locally: the arm does quite well for the purpose.

the late Dr. Hillier, who says (in his Monograph on Children's Diseases) that from experience he had been led to abandon mercurial treatment for Pleurisy;[1] and I believe that, whatever some of the class-books may still say, mercury is practically given up by the best physicians in this country, not only in children's pleurisy, but in that of adults. It seems the general opinion among those with whom I have conversed, that the absorptive action with which mercury used to be universally credited is more than doubtful in the case of pleuritic effusions, whether fibrinous or serous. And certainly, if it fails to do good, mercury may do very sensible harm. I have seen cases in which it apparently produced the most decided anæmia—at least there was scarcely any other possible cause for the latter condition—which set in rapidly after the first occurrence of ptyalism.[2]

The treatment by so-called "counter-irritants," as pursued by many physicians, is no less repugnant to me than is that by mercury or bleeding. Let me make two admissions. In the first place, the mere application of a mild mustard plaster, or, still better, of a hot poultice, or epithem, undoubtedly may give some ease; perhaps even arrest incipient inflammation; and the use of *small flying* blisters, in the limited attacks of Pleurisy which are so common in phthisis, undoubtedly appears to give relief in many cases. But the use of large blisters, especially if kept open, appears to me both useless and often prejudicial. I shall not repeat here what I have said at length elsewhere;[3] suffice it to say that I adhere to my opinion already stated, which is the same as that previously announced by many of the greatest masters of practical medicine in the present century.

The practice of painting the chest-wall with iodine, though not open to the same positive objections as apply to blistering, has never, in my experience, yielded any very positive results. It is, I believe, very inferior in utility to the application of the simple adhesive or the Burgundy pitch-plaster, to afford mechanical support; this really does sometimes appear to favour absorption of the fluid, and it usually gives much comfort.

The employment of diuretics to promote absorption is another point on which I find myself at issue with the opinions of many. The

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[1] See also Meigs and Pepper on the Diseases of Children.

[2] I cannot help making a digression here on the subject of the supposed absorptive action of mercury on inflammatory lymph. So repeatedly have I seen attempts made, without one particle of success, to induce the absorption of pleuritic, peritonitic, and pericarditic lymph by means of this drug, that I have seriously reflected on what could possibly have given rise to the old unreasoning confidence in its power to act in this way. After the best inquiry possible to me, it seems pretty certain that the only groundwork was the assumption of a necessary analogy between *lymph effused in the iris* and that effused elsewhere. Now, to say nothing of the special relations (unintelligible, no doubt) of mercury to *syphilitic* products, it is certain that mercury possesses a strong physiological predilection for the whole territory innervated by the trigeminal nerve; and I believe that there is something quite peculiar in its action on the nutrition of the eye, the mouth, the nose, and the face, and on the pathological products of inflammation in these parts.

[3] "On the Popular Idea of Counter-irritation," *Lancet*, Feb. 26, 1869; "The Theory of Counter-irritation," *Practitioner*, April 1870.

only drug which has appeared to me, in some cases, directly to promote absorption by means of increased diuresis, is iodide of potassium, in quantities amounting to from 6 to 18 grains daily, according to the age of the patient. I think it is worth trial for two or three days (along with the external use of iodine) when effusion comes to a standstill.

The medicine, however, which stands quite alone in its power to promote the process of absorption is *iron*—best given in the form of the *muriated tincture;* and in all cases where there is marked anæmia it should be exclusively employed from the moment when the necessity for administering opium ceases.

As regards purgative medicines, the utmost that I can recommend is that, if necessary, such mild medicines may be used as may suffice to prevent actual loading of the bowels, which, especially in the case of children,[1] might seriously increase the mechanical distress in the chest. Actual purgation is always mischievous in Pleurisy, although it is sometimes very useful in hydro-thorax.

The use of alcohol is a matter requiring much care and judgment. In primary acute pleurisy it is usually best dispensed with, unless the patient is unable to take other nourishment; in this respect Pleurisy differs much from pneumonia. But in secondary pleurisies stimulants will often be needed, and here the amount of the dose must be ruled, not by any routine, but according to those indications of the pulse, the temperature, and the urine, which I have fully described in my lectures on Acute Diseases at the Royal College of Physicians,[2] and elsewhere.[3]

As regards all other matters in the treatment of secondary pleurisies, it is absolutely necessary that I should leave them to be dealt with by the authors who describe in this "System of Medicine" the various diseases of which Pleurisy is apt to be a complication.

One word must be said about a mode of treatment for Pleurisy which I confess that I have never attempted: I mean the employment, so common on the Continent, of *cold* to the chest, and the use of cool baths. I desire to pronounce no judgment whatever on the matter; but those who wish to know more of the system should study the remarkable statements of Niemeyer,[4] a very trustworthy witness, as to the effects of ice-cold applications to the chest.

Paracentesis Thoracis.—A new era has been inaugurated in the treatment of Pleurisy by the development which the operation of tapping the chest has received within the last few years. There is practically no use in going back further into the history of the operation than about thirty years; previously to this there was no real certainty or agreement as to its use except as a *last resort.* It was Trousseau who first had the acuteness and courage to lay down

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[1] Ziemssen (op. cit.) particularly points this out.
[2] *Lancet*, vol. ii., 1867.
[3] *Practitioner*, "Wines in Acute Disease," August 1870.
[4] Handbuch der Speciellen Pathologie u. Therapie, vol. i.

the proposition that in extensive effusions, whether of serum or pus, we ought not to wait till death is imminent, but operate with the view of warding off the dangerous attacks of orthopnœa which, as he proves by a series of remarkable cases, may unexpectedly seize the patient, and carry him off with great rapidity. Trousseau, however, encountered great opposition, both in his own country and elsewhere, and although some of his brilliant results undoubtedly startled the medical world, it may be doubted whether the operation would not have been relegated, after his death, to its former limited sphere, had it not been for the interposition of a very able and clear-sighted American physician, Dr. Bowditch.[1] This gentleman had long felt the futility and the culpable inefficiency of treatment which allowed patients to suffer the misery and danger involved in the retention for months together of fluid in the pleura, but it was not until the invention, by Dr. Morill Wyman, of his excellent suction instrument, that Dr. Bowditch saw his way to the safe and effective performance of paracentesis on the large scale. From that date (1850) till the present time Dr. Bowditch has performed the operation 250 *times, in* 154 *persons, without once seeing any evil, or even any very distressing symptoms resulting from it;* while, on the other hand, it has saved a large number of lives that must otherwise have been sacrificed. " Surely," as the author remarks, " this amount of experience by any one deserves the attention of the profession." To this I warmly assent, and must add that there appears to me to be no opposition to Dr. Bowditch's views by men who have fairly tried his practice, but only by theorists who are afraid of its imaginary results.

Formerly paracentesis was supposed to have two functions only in Pleurisy : that of averting suffocation which was *actually* impending, and that of letting out collections which were pretty certainly conjectured to consist of pus. But against these advantages were to be set, it was thought, the fact that the fluid would inevitably re-form, and re-form *ad infinitum,* and after very few tappings would become purulent (even if air could be excluded from the pleura, which was held almost impossible), thus surely undermining the patient's constitution. But the great and dreadful danger was that of admitting air into the pleural cavity ; that, it was said, inevitably led, not merely to a continuance or aggravation of the purulent formation, but also to the putrescence of the pus, and the rapid depression of the vital powers under the combined influences of profuse suppuration, the absorption of noxious gases, and often the absorption of matter capable of inducing pyæmia. Tapping was therefore held to be inapplicable to the treatment of a merely serous effusion which did not immediately threaten life from mechanical pressure. This feeling prevailed the more strongly because some of the greatest masters of medicine of the present century had declared that primary Pleurisy, with proper

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[1] Dr. Bowditch's original papers are in American Journ. of Med. Science, April 1852; American Med. Monthly, January 1853; Boston Med. and Surg. Journ., May 1857. See also his final paper before New York Academy, 1870.

medicinal treatment, should never be fatal; while in secondary pleurisies it was felt that an element of uncertainty underlies the whole prognosis, which disinclines the physician for doubtful, and possibly dangerous, modes of treatment.

It can hardly be doubted that the whole feeling about the dangerousness of paracentesis rested upon the use of clumsy and imperfect means of operation, and on exaggerated ideas of the evil effects of admitting a small quantity of air into the pleural sac. With regard to the first point, we are entitled to say that it is quite possible so to operate as to ensure that no damage will be done to viscera, and that no more than a trifling quantity of air will be admitted to the pleura. And upon the second point we may certainly now assure ourselves that there is no reason to fear serious mischief from the admission of a limited quantity of air if the opening made in the operation be afterwards properly closed. It is even unnecessary, as Dr. Bowditch's large experience has shown, to make the opening valvular. But the most important advance that has been made is the invention of apparatus which allows of the operation being made either simply exploratory, or carried on at once to evacuation of the fluid. With the instrument either of Bowditch or of Dieulafoy [1] we introduce a very small trocar and cannula guarded with a tap, and by attaching a suction syringe and opening the tap, we withdraw a small amount of fluid, the exact nature of which we can identify: if we elect to continue the evacuation, we can do so with the aid of the syringe; if, on the other hand, no fluid can be obtained, the guard-tap has prevented the entrance of air, and we can withdraw the cannula and close the wound without having done the least mischief. By the use of the small cannula we are able to operate without risk, because, in the case of an entirely mistaken diagnosis, we should have done no damage, even though we had perforated a consolidated lung, a solid tumour, or an intercostal artery. The suction power of the vacuum-syringe will enable even thick fluid, such as somewhat concentrated pus, to be withdrawn through the smaller-sized cannulæ; but the puncture is such a trifle that, in case of our desiderating a larger tube, the smaller one can be withdrawn, the finger being pressed on the spot as it emerges, and the more capacious cannula introduced at the same place.

The site of puncture should be selected in ordinary cases according to Bowditch's rules:—Find the inferior limit of the sound lung behind, and tap two inches higher than this on the pleuritic side; at a point in a line let fall perpendicularly from the angle of the scapula. Push in the intercostal space here with the point of the finger, and plunge the trocar quickly in at the depressed part; be sure to puncture rapidly and to a sufficient depth, or you may be balked by the false membranes occluding the cannula.

[1] It is right to say that Dr. Protheroe Smith claims, with apparent reason, to have been the actual inventor of the instrument now made by a French instrument maker, and employed by Dr. Dieulafoy.

It will sometimes happen that with the greatest care and trouble we are unable to get a flow of fluid at the point where we first puncture; it is then our duty to try elsewhere, for our failure may be owing to unusual thickness of the false membranes in the lowest inch or two of the pleural cavity. We thereupon repeat the puncture a little higher up, and further towards the axillary line;[1] and here we perhaps find fluid: at any rate, no harm has been done by the two punctures.

The circumstances under which paracentesis ought to be performed for Pleurisy are the following:—

1. In all cases of Pleurisy, at whatever date, where the fluid is so copious as to fill one pleura, and begins to compress the lung of the other side; for in all such cases there is the possibility of sudden and fatal orthopnœa.

2. In all cases of double Pleurisy when the total fluid may be said to occupy a space equal to half the united dimensions of the two pleural cavities.

3. In all cases where, the effusion being large, there have been one or more *fits* of orthopnœa.

4. In all cases where the contained fluid can be suspected to be pus, an exploratory puncture must be made; if purulent, the fluid must be let out.

5. In all cases where a pleuritic effusion occupying as much as half of one pleural cavity has existed so long as one month, and shows no sign of progressive absorption.

The *limits* of the operation form an important question. Formerly one great error seems to have been, that operators were often too anxious to extract the whole of the fluid; in this way they often protracted the operation to a mischievous extent, and gave abundant opportunity for that very entrance of air to the pleura which was theoretically so much to be dreaded. Among the latest writers, Bowditch and Murchison[2] have most authoritatively shown that it is neither necessary nor useful to extract the whole of the fluid, and that the removal of just so much as may be necessary to relieve substantially the mechanical distress, will in most cases give the necessary spur to the natural process of absorption by means of which the rest of the fluid will be taken up. One rule seems absolute : the withdrawal of fluid must be arrested the moment that the patient begins to complain of constricting pain in the chest or epigastrium. Even in the case of purulent effusion there can be little doubt that absorption often takes place, though unquestionably there is here a danger that concrete cheesy matter may be left unabsorbed, and under unfavourable circumstances may become the starting-point of tubercular infection.

The case of Pleurisy in *children*, as regards paracentesis, requires special consideration. There can be no doubt that in young subjects

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[1] Bowditch, last pamphlet, 1870.
[2] Lancet, 1870, vol. i. p. 221.

there are physical and vital reasons which might lead one to hope more strongly for complete recovery by natural means than we could do in the case of adults. The softness of the lymph exuded is proportionably greater than in later life, and it is comparatively rare to find adhesions so strong as to bind the lung down with a firmness which renders subsequent expansion impossible; and, unquestionably, the vital activity of the processes of absorption is greatest in early life. But, on the other hand, there is a much greater tendency of effused serum to take on a purulent character in children than in adults; and the dangers of a long-retained purulent effusion are now seen to be much more formidable in presence of recent investigations as to the artificial generation of tuberculosis than they formerly appeared. This latter view of the case has been painfully impressed on my mind by a succession of cases, three in number, in all of which empyema has preceded and apparently caused tuberculosis in children who were, individually, remarkably well formed and robust. Two of the patients belonged to families in which there was a taint of phthisis, the other to a family perfectly free, for at least two generations, from any such disease. And seeing that there is in children a greater possibility of rapid re-expansion of the lung (both on account of the vigour of their respiratory efforts and the relative weakness of the fibrinous adhesions), we may the more reasonably hope that the removal of the whole or a portion of the liquid will be followed by a favourable turn in the progress of the disease. I regard as a typical instance of judicious and successful treatment the case recorded by Dr. Murchison (Lancet, loc. cit.), in which a boy of seven years was tapped on the twelfth day from the initial shivering and attack of pain, and twenty-four ounces of clear serum were withdrawn. Only two days were spent in therapeutic experiments after his admission into the hospital; and as these were without effect, and the effusion was large, displacing the heart and causing some (though not great) dyspnoea and weakness of pulse, the operation was done. Only part of the fluid was withdrawn, and, notwithstanding precautions, some air entered; but the case did perfectly well, and in one month more recovery was substantially complete. The only thing lacking in this case, according to my thinking, is, that the vacuum instrument of Bowditch or of Dieulafoy (Protheroe Smith) should have been employed; the discharge-pipe terminating in an india-rubber tube which dipped under water (*ex abundante cautelâ*).

The following statistics of the operation on children, as gathered from the hospital service of M. Barthez, are reported by Verliac.[1] Thoracentesis was performed on nineteen patients:—1. Simple acute Pleurisy, two cases; simple puncture, cure of both without reproduction of the liquid. 2. Serous tubercular Pleurisy, two cases; simple puncture, one cure without reproduction of the liquid, one death from convulsions six days after the operation. (It is not likely that the latter caused the fatal attack.) 3. Pleurisy symptomatic of heart-

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[1] Op. cit. p. 107.

disease and vascular compression, one case ; cure of Pleurisy after six punctures.   4. Pleurisy with purulent effusion, twelve cases; five cures, seven deaths.

Let me add to this the statistics collected by M. Guinier[1] of Montpellier, of 31 cases of children tapped by himself and others.   The patients were of all ages up to 14 ; as many as 16 were in their seventh, eighth, or ninth year.   In one case the operation cured a large sero-purulent effusion in a sucking child *twelve months old*.   There were six times as many successes as failures, and the mortality was not in the ratio of the age.   The operation itself *never* seemed to do any harm ; in every case much immediate relief was obtained, and in the few fatal cases the operation never seemed to accelerate, but rather to retard the advent of death.

I must cite, also, the valuable authority of Hillier for operation in Pleurisy in children ; if done early, he says, it is not dangerous.[2]

Among other highly respected names, may be quoted Dr. Gairdner, of Glasgow,[3] Dr. J. W. Begbie,[4] and Dr. Fraser,[5] who have had the courage to follow out the more extended application of thoracentesis which Dr. Bowditch has inaugurated.   Personally I have been so far unfortunate that I have scarcely had any opportunities for employing the improved vacuum instruments since I became acquainted with them : although I have witnessed their results in the hands of others.   But I was a believer in the need for more extended use of the operation long before I chanced to hear of Bowditch's discovery : and in two cases, as far back as 1862 and 1863, I tapped with the distinct intention of withdrawing a part, only, of a serous effusion (of four and six weeks' date respectively), and employed no other precaution than that of making the opening valvular.   I did not conduct the liquid under water, but merely guarded the orifice of the cannula with the thumb the moment the stream showed signs of interruption, took much pains in withdrawing the cannula without unnecessary admission of air, and immediately well closed the external wound.   No doubt some air entered, but no harm was done; both patients steadily recovered without reproduction of the fluid.   One was a girl aged 17, otherwise healthy ; the other a lad of 12, singularly bright and precocious, but with a dangerously suggestive family history.

I have thought it pardonable, and even necessary, to devote a somewhat large proportion of this article to the question of paracentesis, because I believe it is the duty of the writer on Pleurisy, in a " System of Medicine " published at the present day, to speak with no uncertain sound on this question ; and in order to command the confidence of readers, it has been necessary to show the manifest tendency of a large number of the best practical men of the day.   Fortified by the evidence above cited, and by the remembrance of a great deal more

[1] Bull. de l'Acad. de Médecine, tome xxx p. 645.
[2] Brit. Med. Journal, Aug. 3, 1867.   [3] Clinical Medicine, 1862.
[4] Edin. Med. Journal, June 1866.   [5] London Hospital Reports, 1865.

that could be produced, I venture to say, decidedly, that practitioners must throw aside the timid and vacillating rules of conduct which the majority of the text-books still prescribe. Tapping is not to be looked at as a dangerous last resort, appropriate only to a few cases. It must become an every-day remedy for cases where an effusion, purulent or not, lingers for more than a very limited period : for the operation may be so conducted as to be perfectly harmless, while no one who knows the facts of recent pathology dare say that even a serous effusion will remain harmless, still less a purulent one. It remains to say a few words on the treatment of those least fortunate cases where, from one cause or another, a purulent fluid forms and re-forms with great rapidity after each tapping, and perhaps becomes putrid and stinking. Where it is only a question of excessive purulent secretion, simple washing out of the pleura with warm water after tapping may possibly change the action of the membrane, but in most cases it will be necessary to keep the cannula in, cork it up, and daily allow the exit of pus, and then wash out the cavity. But in my opinion, if it comes to this, the better plan by far is the drainage tube. A needle-eyed probe, being introduced through the original opening, is carried through to the opposite chest-walls, and is there made to protrude the muscle and skin of an intercostal space, the finger outside carefully feeling for it. The probe is cut down upon, forced out through the chest-wall, and threaded with a strong thread ; this is then drawn back through the chest till it comes out at the original opening. The thread is fastened to an india-rubber drainage tube (pierced with openings in the manner devised by Chassaignac), and the latter is then drawn through the chest till it issues through both orifices. Nothing more then remains but to tie the ends of the tube lightly together.

The use of iodine injections need not, I think, be recommended, save in cases of fetid purulent secretion : in this I agree with the opinions of Guinier, Fraser, and other high authorities. The solution should be one part tincture of iodine to four of tepid water. There is abundant evidence that even a long course of such injections does no harm, and it often appears to do good. Possibly the iodine injections may alternately be altogether superseded by the use of weak carbolic acid solutions. The combined use of disinfectant injections and the drainage tube has proved successful in a good many cases apparently of the worst augury; even, for example, in putrid empyema, secondary to puerperal fever.[1]

I shall sum up the treatment of Pleurisy in a few words. The pain must be met by opium or morphia (preferably injected), by hot poultices, and abstinence from movement (at a later stage the side may be supported by stout adhesive plaster for the same purpose). Acetate of ammonia or acetate of methylamine may be given—not in doses to produce profuse *sweating*—but in moderate *stimulant* doses.

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[1] See Kussmaul (Deutsches Archiv für Klin. Med. iv. 1868) for some interesting recoveries after paracentesis and disinfection of stinking fluids.

The diet should be highly nourishing, but carefully adapted to the state of digestion. The bowels should be kept from actual loading, but no *purgation* should be attempted. The only diuretic worth trying in the stage of fixed effusion is iodide of potassium in small doses; and if this fails, it is best at once to have recourse to muriate of iron. But if at the end of fourteen to twenty days for a child, or three weeks *to* a month for an adult, from the initial symptoms, the fluid does not show real signs of diminution, paracentesis should be performed: and this rule is absolute, both for primary and secondary pleurisies, except where the case is hopeless on other grounds.

HYDROTHORAX.

By Francis E. Anstie, M.D., F.R.C.P.

DEFINITION.—Passive non-inflammatory effusion of serum, due either to mechanical obstruction of circulation, or to blood-poisoning.

HISTORY.—The history of Hydrothorax really constitutes a part of the history of the various organic and constitutional diseases of which it is a mere episode. It was once the custom to speak of this malady as if it were a variety of pleurisy; in reality there is a broad distinction between the two affections. We shall discuss the points in which they approximate under the heading of Pathology; meantime, we may say that their history is essentially different. Hydrothorax, properly so called, lacks several of the most important "notes" of inflammation. It arises, without febrile disturbance, in the later stages of disorders which either mechanically embarrass the circulation through the chest, or alter the specific gravity and the chemical relations of the blood-serum, or do both these things, so as to promote a purely physical exosmosis. It is thus often due to diseases of the heart, particularly those of the right side, and it is not a very infrequent result of renal disease; but in perhaps the majority of cases a combination of renal and cardiac mischief is the cause. The course of Hydrothorax is eminently chronic, and the disease is often entirely intractable; in fact, Hydrothorax occurs in many cases only as a part of the closing scene of chronic organic disease.

SYMPTOMS.—The invasion of Hydrothorax is usually stealthy and unnoticed, there is no febrile movement, and the only noticeable matter is the steady increase of dyspnœa. At last, and sometimes after a day or two only, the patient is in a state of gasping orthopnœa, with livid lips and the greatest appearance of distress; he is quite unable to lie down. Then, on examination, we find the physical signs of fluid in both pleuræ; it may be also in the pericardium. The effusion being bilateral, we find no displacement of the heart; but the diaphragm is nearly always pushed downwards, sometimes very greatly. When the effusion is large, the embarrassment of the heart is shown by the small and feeble pulse.

PATHOLOGY.—The nature of the effusion in Hydrothorax may vary within rather wide limits; but it usually contains far less albuminous and fibrinous material, and far fewer cells (whether of epithelium, or white blood-corpuscles) than are found in pleuritic effusions. It may even be doubted whether the fluid of a passive effusion contains any blood-corpuscles at all; but from the readiness with which a clear Hydrothorax serum sometimes converts itself, if air be admitted to the chest, into pus, the presence of blood-corpuscles would appear probable.[1] When death has taken place without any puncture having been made, the pleura is found free from all lamellar fibrinous deposit, and the lung is simply compressed, not bound down by adhesions.

DIAGNOSIS.—The distinction of this affection from real pleurisy is not always easy; but in most cases the history points strongly to the true nature of the effusion. The simultaneous occurrence of other dropsies, together with the absence of initial fever, enable us, usually, to say that Hydrothorax and not pleurisy is present: but on the one hand there may be no distinct dropsy anywhere but in the chest; and, on the other hand, true pleurisy may sometimes (*e.g.* after scarlatina) coincide with anasarca. Acute rheumatism supervening on old cardiac, or cardiac and renal disease, sometimes presents signs of a double pleural effusion, the nature of which it is difficult to decide; especially as the greatest pallor and depression in such cases may coincide equally with a pleurisy or a Hydrothorax. Notwithstanding these occasional difficulties, however, it is usually possible to give a tolerably decided diagnosis from a comparison of the history and the clinical features of the disease.

PROGNOSIS.—How bad this is will be evident from the circumstances of great bodily depression in which Hydrothorax always arises, and from the necessarily more or less constant operation of the cause of dropsy, tending to a continual reproduction of the fluid even if we have been fortunate enough to witness its reduction or removal. Nevertheless there is great room for bold and intelligent treatment in a certain percentage of cases of Hydrothorax; and recoveries sometimes take place in a surprising manner. Many patients have had weeks, months, or even a few years, added to their lives in this way.

TREATMENT.—The tendency of the best modern practice in regard to Hydrothorax may be said to be nearly the reverse of that with regard to pleurisy. The operation of paracentesis is rarely applicable: it should be reserved almost exclusively for the prevention of threatened asphyxia when both pleuræ fill rapidly to a great height. On the other hand, the effect of diuretics, and still more of

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[1] I tried to convince myself on this point, some time since, by microscopic examination of a typical hydrothorax fluid; but could not make up my mind upon the matter. Dr. Walshe speaks of "pus-cells" as being present.

hydragogue purgatives, is often most striking. Of the former, infusion of digitalis in half-ounce doses, with thirty grains of bitartrate of potash twice or three times daily, has yielded me better results than any other. Of purgatives I only recommend one, viz. elaterium, which is incomparably superior, in my opinion, to all others. Great care ought to be taken to select a first-rate specimen of the drug, and then (diuretics having been fairly tried first) we need not scruple to use the elaterium boldly. One-fourth of a grain may be given (combined with a little hyoscyamus), and repeated in four hours ; very usually two, or at most three doses will suffice to produce a very copious watery catharsis. It might be thought that this would kill such feeble creatures as Hydrothorax patients generally are, but if care be taken to give a little stimulant at the time that the bowels act, the effect is very far from exhaustive; the rapidity with which the fluid diminishes in the chest, and the consequent relief to all the patient's sensations, in favourable cases, must be seen to be believed. The moment that a decided impression has been produced, either by diuretics or by purgation, we must begin the use of muriate of iron, in twenty-drop doses of the tincture four or five times in the twenty-four hours : in this way we secure the best chance open to us of preventing the re-accumulation of the fluid.

Do what we will, however, it is of course inevitable that a majority of our patients will succumb : and those whom we for the moment cure of Hydrothorax are only temporarily relieved from danger.

PNEUMO-THORAX.

By Francis E. Anstie, M.D., F.R.C.P.

Definition.—Accumulation of atmospheric air, or other gas, in the pleura.

Varieties.—I. Non-perforative. Collection of gas (*a*) from decomposition in gangrene of the pleura; (*b*) from decomposition of an ordinary pleuritic fluid; (*c*) air replacing sero-purulent fluid, suddenly absorbed; (*d*) secretion of air by pleura.

II. Perforative. (*a*) Surgical, from penetrating wounds of thorax, or fractured ribs lacerating the lung, or violent contusion tearing the lung. (*b*) Perforation of lung and pulmonary pleura, from disease in the lung: (1) Tubercular, (2) gangrenous, (3) diffuse pulmonary apoplexy, (4) hydatids, (5) cancer, (6) emphysema, (7) abscess, (8) rupture in hooping-cough. (*c*) Perforation of lung from without: (1) by disease of bronchial glands, opening into pleura and bronchi, (2) by emphysema, (3) by parietal abscess. (*d*) Rupture of œsophagus opening into pleura.

This formidable-looking list of possible varieties of Pneumo-thorax simplifies itself greatly when looked at from the practical physician's point of view. We may usefully abstain from special consideration of the non-perforative kinds altogether, from their great rarity. Of the perforative kinds, we put aside the surgical varieties, as not coming within the scope of this work. Of the remaining varieties of perforative Pneumo-thorax, all, save one, are individually so rare as to deserve little more than the bare record of their occasional occurrence. More than 90 per cent. of perforative cases from disease of the lung itself are, according to Walshe, "tuberculous" (*i.e.* produced by some form of phthisical lung-disease), and, in fact, the subject of Pneumo-thorax, from the physician's standpoint, falls almost entirely under the domain of phthisis.

Clinical History.—The typical access of Pneumo-thorax is distinguished by the sudden occurrence of sharp pain in the side, and intense dyspnœa of the most distressing kind; occasionally, besides these, there is the distinct sensation, at the moment, of tearing inside

the chest, followed by a feeling as if fluid trickled or poured down the side. Collapse, with coldness of the surface and cold sweat, is present in the majority of cases.

But the symptoms by no means always take this striking form: there are cases in which neither pain nor dyspnœa are present at first in at all a high degree; and there are many more in which, after the first moments of severe suffering, the patient enjoys comparative repose until the secondary symptoms, viz. those of pleural inflammation, set in; and this sometimes represents a considerable period of comparative pause. But the inflammatory process invariably, and for the most part very speedily and severely, sets in: and often there is again very rapid breathing before the recurrence of great conscious distress. In fact, *rapidity* of breathing is almost a physical necessity from the moment of the rupture, and it is great, not merely absolutely, but relatively to the pulse frequency, though the latter is also very much augmented. In the worst cases there is never one minute's cessation, from the moment of the catastrophe till death, of the most acute pain and the most distressing orthopnœa; this was exemplified in a little boy who was under my care at the Belgrave Hospital for children about four years ago.

The physical signs of Pneumo-thorax give a very decided answer to our suspicions as to the nature of the case. The chest is very much, the affected side almost altogether (especially at the lower part), debarred from movement, the breathing is carried on mainly by the abdominal muscles; if the affected side moves at all evidently, the intercostal spaces are seen to be greatly depressed. Percussion gives out at first a merely much louder sound, with a graver pitch than in health; as the distension increases it becomes quite drum-like, and, if distension reaches the very highest grade, it becomes dull and muffled again—a well-known phenomenon of extreme air-tension. Occasionally percussion gives out an amphoric note. Palpation discovers weakening or abolition of vocal fremitus. Auscultation detects either great enfeeblement or complete suppression of the breath-cough, and voice-sounds, according to the amount of air in the pleura; the heart-sounds are either greatly weakened, or, occasionally, they are transmitted with a metallic ring.

As the distension proceeds the case becomes the more unmistakeable; the mediastinum, heart, and diaphragm are notably displaced, and the tympanitic percussion-sound is heard to extend continuously even beyond the further sternal border.

When fluid becomes effused to a notable extent there are of course the signs, described under pleurisy, of a liquid effusion in the lower part of the chest, together with the signs above mentioned, of air in the chest. There is also, when the fluid reaches any considerable amount, easily detectable fluctuation when the patient is shaken; and more occasionally and variably we can thus produce the true *splash*, with *metallic ring*. Moreover, with rare exceptions, the fluid demonstrably changes its position with changes of the patient's

posture: in this respect Pneumo-thorax assimilates to hydrothorax rather than to pleurisy. At the boundary line between fluid and air there may be amphoric percussion-note, and a vibratile sensation communicated to the fingers. The displacement of viscera reaches, in bad cases of hydro-pneumo-thorax, the extreme degree which is ever observed.

DIAGNOSIS.—The only affection with which Pneumo-thorax can possibly be confounded is extreme emphysema; but there cannot be more than a momentary difficulty, even here. Emphysema must be most unusually pronounced before the percussion-note reaches any-thing like the tone of that heard in Pneumo-thorax; but then such emphysema is always symmetrical, while Pneumo-thorax affects only one side. But, indeed, the whole aspect of the two affections is quite different.

PROGNOSIS.—The prognosis of Pneumo-thorax is, on the whole, very bad, especially during the first day or two; if the patient survives for two or three days, his chances have materially improved. The great majority of fatal cases die within a week, and of these the largest part within the first two days. By common consent of autho-rities, however, there is a great uncertainty in the matter: cases which appear comparatively slight at first sometimes terminating fatally in a few days, while others, which at the outset seemed desperate, go on steadily improving, and regain comparative health; usually, however, they retain the signs of air and fluid in the pleura. In a few cases an absolute cure takes place; these are mostly instances either of traumatic Pneumo-thorax, or else of empyema discharging itself through the bronchi. A few cases, however, even of phthisical Pneumo-thorax do recover; the opening becoming closed by lymph, and the air and fluid getting partly or wholly re-absorbed. A variety of Pneumo-thorax from which striking recoveries have taken place is that in which the rupture has been more the consequence of great muscular exertion than of any severely diseased condition of the lung. Such are some of the cases where the rupture has taken place during the paroxysms of hooping-cough; and a remarkable instance of an analogous kind has been reported by A. Vogel.[1] An unmarried woman, aged twenty-nine, who had borne ten children, had acted as a wet-nurse for a long time after each confinement, and had been perfectly well except that recently she had suffered from catarrh and an obstinate cough, in the midst of a sudden muscu-lar exertion felt a sharp pain in the right side, and was seized with the most intense dyspnœa. When seen some hours later there were all the signs of the most complete Pneumo-thorax of the right side, with great displacement of the heart, lung, liver, &c., and severe collapse. Opium gave temporary ease, but on the next morning the anguish returned with waking, together with vomiting and choking

[1] Deutsches Archiv für klin. Med. ii. p. 244, 1866.

sensations; yet no sign of pleuritic exudation could be detected. Morphia again gave relief to the pain and dyspnœa, and from this time all the symptoms speedily declined. In four days from the attack the patient had entirely recovered, and when seen a year later not a single trace of any mischief could be detected.

Of late years, indeed, a great deal of evidence has been collected to show that the mere influence of air upon a healthy pleura is extremely slight, and scarcely predisposes to inflammation at all: this comes out remarkably in the experiments and observations of Demarquay.[1] The same observer has also shown that a gradually decomposing collection of gas in the pleura is likewise harmless, except where sulphuretted hydrogen or sulphide of ammonium is developed. On the whole, it can scarcely be doubted that the larger part of the influences which determine the fate of patients with Pneumo-thorax depends upon unknown vital differences which there is little probability of our ever being able to estimate beforehand.

TREATMENT.—The treatment of Pneumo-thorax is, necessarily, entirely palliative, and directed to the object of enabling the patient to survive the intensely depressing influence of the first shock, and that of the subsequent inflammation. The first step to be taken is the hypodermic injection of a full dose (half-grain) of morphia; and this medication may be administered twice, or in exceptional cases three times a day during the first two or three days, the hope being that it may possibly avert the threatened pleuritic inflammation. Dry cupping to the chest, frequently repeated, has been said to give very great relief in many cases. I cannot approve either of bloodletting, in any form, or of mercury; for the phthisical cases they are directly injurious, and in any case hypodermic morphia is likely to effect all the good which either of these remedies could be supposed capable of producing. Hot poultices to the chest undoubtedly give ease; they should be continually renewed. The great depression which is felt can, I think, generally be more suitably met by the internal administration of ℨss doses of ether, every three or four hours, than by alcoholic stimulants, though the latter are sometimes absolutely necessary.[2] If the patient survives the first few days, it will be proper to administer mineral acid and bark, cod-liver oil, or muriate of iron. And throughout the illness the greatest pains must constantly be taken to maintain the strength by easily digestible nutriment; and if the stomach be too irritable to bear this well, nutritive enemata must be unhesitatingly resorted to.

The question of paracentesis may be suggested by the extreme distress of respiration. In the phthisical cases, which form the large majority of those which the physician has to treat, this step could only be regarded as a very temporary palliative, and accordingly should only

[1] Gaz. Médicale, 32, 1865.
[2] I have seen cases in which alcoholic stimulants apparently much increased the acute pain. On the other hand, they occasionally do striking good.

be employed as a last resort to procure a temporary respite when some very important object is to be secured by keeping the patient alive a little longer. It might be far more justifiable in cases where we had strong reason to suppose that the rupture was mainly accidental, and that the lung was free from serious internal disease. But I am not aware that any considerable statistics exist which might guide us to a conclusion on such a very doubtful point.

APPENDIX TO SIR WILLIAM JENNER'S ARTICLE ON EMPHYSEMA OF THE LUNGS.

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WAXY LIVER.

By J. Warburton Begbie, M.D., F.R.C.P.E.

The expression Waxy is now almost universally understood to indicate a disease of the Liver attended by great enlargement; not resulting from, or having any intimate connexion with, fatty degeneration of the organ, as was at one time supposed, but due to the presence in the liver structure of a peculiar substance, giving it a somewhat translucent appearance, resembling bees'-wax or Canada balsam, and which, whatever be its real nature, as yet undetermined, is readily distinguished from fat.

Rokitansky was the earliest to point out the essential characters of this form of disease, and the earliest also to indicate the very important relation in which it stands to certain cachectic conditions and constitutional disorders: this he did in 1842.[1] Afterwards, through the careful observations of Dr. Gairdner in reference to the Kidney (1848), and of Dr. Handfield Jones and Dr. Sanders, on the Spleen (1852), the peculiar morbid condition in question became more generally recognised. Since that time, our knowledge of waxy degeneration as affecting the liver, spleen, and kidneys, as well as other organs and tissues of the body, has been greatly advanced by the researches of Meckel, Virchow, Frerichs, and several other pathologists on the Continent, and by many observers also in our own country.

SYNONYMS.—Lardaceous, Baconlike, or Amyloid Liver; Scrofulous Enlargement of the Liver; Colloid Infiltration; Die Wachsartige; Speckige oder Amyloide Degeneration der Leber; Wachsleber; Die Speckleber.

ANATOMICAL DESCRIPTION.—The first character of the Waxy Liver to attract notice, is its size. Thus affected, the organ may become

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[1] Rokitansky had himself used the term "waxy" in the sense of a variety of fatty liver, and as something different from the lardaceous (*speckig*, or baconry liver) degeneration. "The waxy," he remarks, "is a variety of the fatty liver. It is distinguished from the latter by a colour resembling that of bees'-wax, by its greater consistence, dryness, and brittleness; and these qualities depend upon a peculiar modification of the infiltrated fat, which, although accumulated to a considerable amount, leaves but few traces on the scalpel."—*Pathological Anatomy*, Sydenham Society's Edition, vol. ii. p. 121.

enormously enlarged. Dr. Murchison remarks: "I have known the liver of an adult affected with this disease weigh upwards of 180 instead of 50 or 60 ounces, and the liver, of which I show you here a portion, weighed one-seventh, instead of a twenty-fifth, of the entire body of the child from whom it was taken."[1] There is only one other form of disease of the liver, which is capable of determining a greater degree of enlargement than the Waxy, and that is the Cancerous. In the slighter degrees of the degeneration, the size of the liver may be little altered, it may even be diminished.[2] Besides increase in size there is augmented density, the organ becomes very heavy, acquiring a peculiar doughy firmness.[3] The capsule is smooth and tense, and the form of the liver little changed. The absence of any change in form must, however, be understood as applying to the liver affected by the purely waxy degeneration. When this is associated with fatty change, the edges are blunt and rounded; when combined with cirrhosis (granular induration), the surface is covered with large and small knobs; and finally, when cicatrices the result of syphilitic disease of the liver remain, there is the formation of irregular lobes, and these may be separated by deep fissures. A knowledge of these circumstances is of importance in the endeavour to form a correct diagnosis, for otherwise the enlargement of the liver, attended by irregularities of surface, might be taken for cancerous disease of the organ. The Waxy Liver, when the degeneration is well marked, presents a pale or fawn-coloured aspect, or, owing to some degree of congestion, there may be a mottled appearance. When cut with a sharp knife, the section is smooth, dry, and firm, while little or no stain is communicated to the blade as in fatty liver.

Waxy disease of the liver has its commencement either in the secreting cells, or in the minute branches of the arteries of the organ.[4] The earliest noticeable change consists in the central portions of the acini, assuming a reddish-yellow colour, becoming at the same time pellucid and firmer than natural, as well as sharply limited from the surrounding dull-grey edge, so that the lobular structure of the organ is very clearly marked. When solution of iodine is applied to the glistening pellucid spots, these become of a deep-red colour,

[1] Clinical Lectures on Diseases of the Liver, p. 23.
[2] "Unter 23 Fällen fand ich drei mit normalem, drei mit verkleinertem Umfang der Leber."—FRERICHS, *Klinik der Leberkrankheiten*, Zweiter Band, 167.
[3] "Die Consistenz von einer eigenthümlich teigichten Festigkeit."—FRERICHS.
[4] That the waxy degeneration has its commencement in the minute arteries of those organs and tissues which become affected by it, is now a well-established pathological doctrine. The first indication, probably, of the participation of the blood-vessels in waxy degeneration, was given by Dr. Gairdner; and among the earliest to make the discovery that, in the kidney, this morbid process has its *commencement* in the minute arteries and capillaries, was Dr. Kirk, the distinguished physician and naturalist, who accompanied Dr. Livingstone on the Zambesi Expedition, and who is now the British Consul at Zanzibar. (See an interesting historical sketch of waxy or amyloid degeneration in the Edinburgh Medical Journal for March 1868.) Virchow observes: "Am schönsten kann man diese Veränderungen verfolgen an denjenigen Gebilden, welche überhaupt als der häufigste Sitz dieser Veränderung betrachtet werden müssen, namlich an *den Kleinsten Arterien.*"—*Die Cellularpathologie*, S. 335.

while the surrounding border is of a pale yellow. As the waxy degeneration becomes more marked, the appearances now described are seen to characterise the entire lobular structure; the lobules become larger, with ultimate obliteration of their borders, and the parenchyma of the gland offers a homogeneous, smooth yellowish red, somewhat glassy surface, in which the open mouths of the blood-vessels are seen, and from these a little thin blood is found to be flowing. Portions of liver thus affected, resemble, according to Frerichs, whose description we have been closely following, delicate slices of smoked salmon.[1] When attention is paid to the progress of waxy degeneration in the individual cells, it is noticed that the fine granular contents of the normal cells by degrees disappear, and a homogeneous clear substance occupies their cavities. In a few cells the nucleus may still be recognised, swollen and lustrous; but in most, the nucleus can no longer be detected. Cells thus degenerated are firmly connected to each other, and sometimes present solid masses in which neither cell, wall, nor areolar connective tissue can be distinguished. In advanced waxy degeneration angular fragments, resulting from the disintegration of the degenerated cells, are here and there to be met with.

Similar changes to those now described as occurring in the secreting elements of the gland take place in the smaller vessels, the walls of which become thickened, unbending, homogeneous, and lustrous, their channel being at the same time narrowed and not unfrequently entirely occluded; thus circumstanced, a vessel has the appearance of a colourless cylinder in which no fine structure can any longer be seen. Considering the peculiar vascular arrangement which exists in the liver, the question occurs, In which of the minute vessels are the changes now described discoverable? Acknowledging the difficulty of determining in every case to which system the affected vessels belong, Frerichs considers it an ascertained fact that generally it is the minute branches of the hepatic artery which are implicated. The morbid change, however, is not confined to them, for Frerichs and others have noticed the same in the portal and hepatic veins.[2] When the waxy degeneration has its seat in the liver cells, the blood-vessels, or connective tissue, a mahogany-red colour is observed when the solution of iodine, as already stated, is brought in contact with the affected portion of the organ; and if a little sulphuric acid is cautiously applied to the surface already moistened by the iodine, the deep-red is converted into a dirty violet, or it may be a blue colour. The latter reaction, however, is rare in the liver.[3]

Waxy degeneration of the liver is not unfrequently associated with fatty deposit in the organ, and also with cirrhosis. The important connexion of waxy disease with syphilis, prepares us to meet with

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[1] Op. cit. p. 167.

[2] "Ich sah inders auch mehrfach erkrankte Capillaren, welche der Lokalität nach der Pfortader und den Lebervenen anzugehören schienen."—FRERICHS.

[3] "Im Allgemeinen sieht man die blaue Färbung der Wachsartigen Substanz in der Leber seltener."—FRERICHS.

those cicatrices and gummy nodules, as well as irregular hepatic lobes which plainly indicate the previous occurrence of the constitutional disorder.

ETIOLOGICAL CONSIDERATIONS.—Waxy degeneration of the liver is more commonly met with in males than in females. Frerichs has collected sixty-eight cases, of which number twenty-three had fallen under his own observation; and of the sixty-eight, fifty-three occurred in males and only fifteen in females. Waxy degeneration affecting the liver appears to be of most frequent occurrence during the period of adolescence. In the sixty-eight instances referred to, Frerichs met with three under 10 years of age, nineteen between 10 and 20, nineteen between 20 and 30, eighteen between 30 and 50, and only nine between 50 and 70.

Reference has already been made to the important observation of Rokitansky, connecting the waxy degeneration of the liver with the previous existence of a depraved habit of body or cachexia.[1] Of this nature there rank in the highest order of importance, diseases of the bones, caries and necrosis, and constitutional syphilis, also syphilis associated with mercurial cachexia. Dr. Graves had noticed the connexion of "hypertrophy of the liver" with syphilis and abuse of mercury. After detailing the features of a case which had fallen under his observation, this distinguished writer proceeds: " Here, then, we have venereal, abuse of mercury, periostitic inflammation, abuse of mercury followed by exacerbation of the periostitis and establishment of mercurial cachexy; and the history of the case is wound up with hypertrophy of the liver. This was the first case in which I had observed this concatenation of diseases ; since that period, I have seen a similar train of morbid phenomena twice in private practice, and once in hospital. First, we have abuse of mercury, then periostitic inflammation and mercurial cachexy, and the scene is closed by morbid enlargement of the liver. Now, I do not look upon this sequence as merely fortuitous. The diseased actions are, I think, related as cause and effect, and each successive condition is consequent on the previous one."[2] It will scarcely admit of doubt that the enlargement of the liver described in this passage by Dr. Graves, is identical with the waxy degeneration, and accordingly Dr. Budd is justified when he remarks that the fact of *enlargement* of the liver being apt to occur in persons whose health is broken down from the combined effects of mercury and syphilis, was first distinctly noticed by Dr. Graves.[3] Frerichs, however, believes that Waxy Liver has been incorrectly imputed to the use of mercury, and he enters a decided protest against

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[1] "This affection," remarks Rokitansky, "is found concurrent with constitutional disease of the vegetative system, especially with scrofulous and rickety disease, with syphilitic and mercurial cachexia, and it may consequently be congenital. It appears that it is occasionally developed as a sequela of intermittent fever in cachectic subjects." —Op. cit. vol. ii. p. 121.

[2] Clinical Medicine, Lecture xxx.

[3] Diseases of the Liver, p. 330.

the views of Graves and Budd, which attaches great importance to the
mercury, asserting that he knows no case where an increase of size of
the liver, and still less where a distinct waxy degeneration of the
organ, has been caused by mercury. Next in order of importance
to syphilis, ranks chronic purulent discharge, and especially such as is
found in connexion with disease of bone or joints.

The influence of intermittent fever in giving rise to waxy degenera-
tion is doubtful, although Rokitansky places the latter as an occasional
sequela of the former in cachectic subjects. Dr. Budd remarks that
he has met with one instance in which severe and long-continued ague
in a boy was followed by scrofulous disease of the glands of the neck
and of the bones, and subsequently by great enlargement of the liver
and ascites. This enlargement Dr. Budd ascribes to the scrofula and
not to the ague, and he asserts that the liver very seldom gets much
enlarged from ague, in this respect offering a notable contrast to the
spleen.[1] The relation of tubercular disease of the lungs and the bowels
to waxy degeneration of the liver is unquestionably far less intimate
than that of fatty degeneration; nevertheless such relationship exists.
Meckel pointed it out, and Wilks and Friedreich as well as Frerichs
have noted it. Dr. Murchison mentions that of fifty-two persons
dying from tubercle and whose autopsies he has recorded, the liver was
fatty in twenty and waxy in six, and in three of the six there was
likewise caries of the bones. In these fifty-two cases, however, the
connexion of tubercle with waxy degeneration was more remarkable
than the occurrence of the latter in the liver alone appears to indicate,
for Dr. Murchison further intimates that fourteen of the entire number
had waxy disease of either the kidneys, the liver, or the spleen.

Besides the causes of waxy degeneration now mentioned, there are
others which must be styled unknown causes, inasmuch as in
certain cases the morbid condition of the liver in question—and the
same remark applies to the degeneration in other organs—appears
independently of any one of the already determined causes.

PATHOLOGY OF WAXY DEGENERATION OF THE LIVER.—This branch of
the inquiry, in its intimate aspect, is still greatly involved in obscurity.
The point of departure in the morbid process, the " primum mobile,"
is as yet undetermined. Frerichs speaks of two possible modes in
which the causes we have already considered may operate : first,
the degeneration may be due to deposition from the blood ; or, second,
the waxy substance may be developed in the part from some albu-
minous matter previously deposited. Virchow, who has devoted great
attention to this subject, insists that the gradual infiltration of the
parts in a waxy degeneration is with a substance brought to them
from without. This view he holds to be greatly strengthened by the
consideration that a whole series of organs is implicated, and that the

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[1] Op. cit. p. 331. The notion that the waxy material is dealkalised fibrine has been
advanced, but this requires confirmation. (See Dickinson, Medico-Chirurgical Trans-
actions, vol. l. p. 55.)

morbid process is not confined to a single spot, but affects at the same time many parts of the body. The only place in which Virchow has observed waxy degeneration apparently independently developed, and not due to changes from without, is permanent cartilage. Although impressed with the belief that the waxy change is determined by deposition from the blood, neither Virchow nor any other observer has hitherto succeeded in detecting any distinct change in the blood.[1]

SYMPTOMATOLOGY.—The enlargement of the liver, in addition to its being great—sometimes so great as to occupy a large portion of the abdomen—is uniform in all directions. The tumour thus formed is dense, firm, and resistant, and its surface is quite smooth. The lower border is usually rounded and free from irregularities. When cirrhosis and syphilitic affection of the liver co-exist with waxy degeneration, irregularities of surface and a lobulated character will be acquired. There is seldom either pain or tenderness, and the patient feels little or no inconvenience from the free handling of the enlarged organ. The extreme size of the liver in waxy degeneration is far from being rapidly attained; on the contrary, many months, and often years, are passed during which a slow and gradual augmentation in its bulk is going on. Dropsy is not a symptom of waxy disease of the liver; therefore ascites, which is so notable a feature in certain hepatic affections, does not occur in it. Neither is jaundice at all frequently met with. Frerichs observed jaundice in two out of twenty-three cases, and in both of these the lymphatic glands in the fissure of the liver were found enlarged. A similar enlargement of the lymphatics, due to waxy degeneration in them, may, by pressure on the trunk of the portal vein, give rise to ascites. Splenic enlargement is of common occurrence, but, unlike the splenic tumour met with in cirrhosis and in cancer of the liver, it is due to waxy degeneration of the spleen, and not, as in these instances, to blood stasis in the organ from interrupted portal circulation. With waxy disease of the liver a similar degeneration of the kidney is frequently associated; and as the latter possesses distinctive characters of its own, it is of much importance to observe the condition of the urine when suspicion attaches to the liver. The urine then of waxy kidney is very generally increased in amount during the greater period of the disease, only becoming reduced in quantity towards its termination.[2] In colour it is pale, in density moderately low (from 1,015 to 1,008), and it contains a considerable amount of albumen. Casts of the tubuli uriniferi are frequently absent from such urine, and when detected in it are usually of the hyaline character. Dr. Murchison remarks that he has never observed the amyloid reaction with iodine and sulphuric acid to be

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[1] Cellularpathologie, S. 339.

[2] Dr. Grainger Stewart, who has specially insisted on the polyuria of waxy degeneration of the kidneys, remarks : "I have never found it absent, except in cases accompanied with severe diarrhœa, or by inflammation of the tubules of the kidneys, or by a peculiar syphilitic deposit in the stroma of the organ."—*A Practical Treatise on Bright's Diseases of the Kidneys*, p. 91.

produced in these casts, but in exceptional cases has found the reaction in some of the cast-off renal cells.[1] The writer has noticed a tendency to the occurrence of epistaxis, sometimes severe in character, to accompany the waxy degeneration when both liver and kidneys have participated in the disease ; he has indeed come to regard waxy degeneration of the kidneys as the form of chronic renal disease with which nasal hæmorrhage, pericarditis, and uræmia are most apt to occur. Where the liver is the seat of this transformation it is then of special importance, in a prognostic point of view, to determine whether or not the kidneys are likewise involved. Vomiting and diarrhœa are of by no means uncommon occurrence in connexion with waxy liver, and for the most part depend on the existence of a similar degeneration affecting the blood-vessels, the villi, and sometimes even the tissue of the mucous membrane itself, of both stomach and intestines.[2] It is in this way that digestion and assimilation are so much interfered with in cases of Waxy Liver, and that the anæmia, which is so characteristic a feature of the disease, is in part at least produced.

PROGRESS AND DURATION.—Waxy degeneration of the liver is essentially a chronic malady, often lasting for a very considerable period— during many months, and sometimes even years. It is, moreover, in the vast majority of cases a fatal disease, and the termination is for the most part due to a gradual process of exhaustion. Not very un-

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[1] In addition to the characters mentioned above, the writer has been led to associate with the passage of an increased amount of pale, lemon-coloured urine, the existence of a very peculiar odour of the breath and of the skin, which, for want of a better term, he has been accustomed to style musty odour. With this odour he has not only in several instances detected the co-existence of the special characters of the urine, dependent on waxy degeneration of the kidney, but has invariably noticed the presence of uroxanthin in greater or less amount. Not only so, but conformably to the experience of Heller, in regard to urines rich in his uroxanthin, the addition of an acid to the urine, or sometimes its continued exposure to the air, has led to the development of a blue colour (indigo-blue, or uroglaucin), or of a notable red colour (urrhodin, indigo-red). It may be conjectured that the pigment which thus finds its way into the urine, is in all probability a product of the transformation of hæmatin. The anæmic appearance of the patient in the advanced stages of waxy kidney is certainly conspicuous. The writer desires to direct attention to the point now mentioned, and while abstaining from directly styling the odour of the breath and cutaneous surface, to which he has adverted, *indigoferous,* he must nevertheless confess that it has frequently struck him as having a pretty close resemblance to the smell which is emitted by musty indigo. On the subject of Iudican, some interesting observations will be found in Dr. Parkes's treatise, "The Composition of the Urine," p. 196.

[2] "Die Schleimhaut von Magen und Darm findet man unter solchen Umständen gewöhnlich dem Anscheine nach wenig verändert, nur blass, durchscheinend und aufgelockert ; bei genauerer Untersuchung ergeben sich indesswichtige Anomalien, besonders in den feineren arteriellen Gefässen, deren Wandungen wachsartig entarten, glänzend, rigide und dick werden, hier und da bis zum Verschluss ihres Lumens. Häufig findet man auch die Substanz der Zotten wachsartig infiltrirt oder es entsteht ein Schwund derselben, durch welchen sie über weite Strecken hin zerstört werden. Mitunter greift die Zerstorung auch auf das Gewebe der Schleimhaut selbst über ; es bilden sich unregelmässige, bis in das submucose Gewebe eindringende Substanzverluste, an deren Rändern man zerfallende schleimhautfetzen bemerkt. Wiederholt sah ich die Kapseln der Peyer'schen und solitären Drüsen vergrössert und von veissgrauer Farbe." — FRERICHS, op. cit., Zweiter Band, 179.

frequently an attack of pneumonia or dysentery, or some other form of abdominal inflammation of a low type, proves the immediate cause of death. Frerichs believes in the possibility of the recently infiltrated waxy substance being absorbed; and such being the case, it is conceivable that a permanent recovery in some cases may occur. What is of special importance, however, to observe is, that the mere fact of the enlargement of the liver being reduced under treatment does not imply recovery. The cachexia with which the enlargement is associated may be firmly seated, and, although a favourable change in the condition of the liver may occur, so firmly seated as gradually to lead to the thorough and complete undermining of the constitution and the inevitable fatal result.

DIAGNOSIS.—From what has already been stated more particularly under the heads of Symptomatology and Etiology, it will be seen that the recognition of Waxy Liver and its distinction during the lifetime of the patient from other forms of hepatic disease, does not involve any special difficulty, at all events when the organ is notably increased in size. The prominent features in diagnosis are, the uniform enlargement and augmented consistence of the liver, with which increased size of the spleen, and very generally also albuminuria of the nature previously described, are associated. If, in addition to these prominent features, there be the tendency to vomiting and diarrhœa and a history of previous syphilis, or of disease of bone, particularly caries, or suppurations, or tubercular affections, the diagnosis may be made with tolerable certainty. Assuredly there is no other form of hepatic enlargement, neither the fatty liver, nor the simple hypertrophy, nor hyperæmic swelling, nor cancerous nor hydatid disease, which, possessing the one similar character of increase in size, do not materially differ in other and these readily observed features.

TREATMENT.—Under this head it is first of all of importance to consider the preventive treatment of Waxy Liver; regarding which Dr. Murchison has truly observed that it has not yet received the attention which it deserves. We have noticed that waxy disease of the liver is apt to occur after long-continued suppuration occurring in any part of the body, and leading to exhausting discharges. It is of importance then to prevent the occurrence of such, or at all events to arrest them when they appear to threaten a lengthened continuance. Syphilis, the intimate relation of which to waxy disease of the liver has been noted, must be met by suitable treatment; and in this case, as well as in that of the other diseases already mentioned, in the course of which the hepatic derangement is apt to be developed, great care should be taken to obviate the occurrence of that cachectic condition of the system which the diseases in question are prone to engender. It is of course in the early stages of the affection that a suitable regulation of diet and the employment of tonic remedies, particularly the mineral acids and the various bitters, are most likely to prove

serviceable; but even in cases which have become marked by enlargement of the liver, the use of the preparations of iodine and iron has undoubtedly proved successful in removing the symptoms of the malady. The iodide of potassium and the iodide of iron appear to have been the remedies chiefly employed and most extensively useful. Frerichs details a case in which the long-continued use of the syrup of the iodide of iron was signally efficacious. Dr. Murchison gives the preference to the tincture of iodine of the British Pharmacopæia, in doses of ten or fifteen minims diluted, three or four times daily. Many of the German physicians, and Dr. Budd in our own country, have employed with marked benefit the hydrochlorate, as well as other salts of ammonia. The writer has witnessed in several instances the remarkable subsidence of both hepatic and splenic enlargements under the use of the muriate of ammonia, in doses of from fifteen to thirty grains, freely diluted, thrice daily. In one instance, and that exhibiting the largest increase in size of the liver which he has ever seen reduced under treatment, the muriate of ammonia in scruple doses was very manifestly the means of effecting the amelioration. In this case there had been previously existing syphilis. When anæmia is notable, the use of one or other of the preparations of iron is called for. Again, vomiting and diarrhœa, which are so apt to occur in connexion with the morbid changes in the stomach and bowels, must be met by such remedies as ice, bismuth, cerium, prussic acid, and naphtha in the former case, and the various astringents, more particularly the sulphuric acid, and the cautious employment of opium in the latter. Diuretics and diaphoretics, rather than drastic purgatives, are to be used when the existence of dropsy adds greatly to the patient's discomfort. The moderate use of alcoholic stimulants is certainly indicated, and the diet should be as nutritious as is consistent with the enfeebled digestive powers of the patient. In the earlier stages of the disease change of air and climate have apparently been serviceable. Frerichs and Wetzlar, as well as other authorities abroad, and some at home, have recommended the baths of Aix-la-Chapelle (thermal, sulphurous, and saline), of Ems, and Weilbach; while the waters which contain the neutral salts in abundance, such as Karlsbad, Vichy, Marienbad, and Kissingen, are much less favourable from the circumstance of their being liable to produce diarrhœa. The writer has known a decidedly favourable influence to be produced by a lengthened sea-voyage, made under circumstances the most suitable as regards diet and other important particulars. And in one remarkable instance of waxy disease, involving apparently to a greater or less extent all the viscera which are liable to its occurrence, and especially characterised by urgent vomiting, the patient expressed himself as only feeling really comfortable when on the sea, remarking that there his sickness is always checked, and that even in rough weather, when most are suffering, he remains quite unaffected.

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END OF VOL. III.

NAME	DATE DUE
J.J. Murick	NOV 26 1935